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TO PROHIBIT SMOKING IN FEDERAL BUILDINGS

(103–16)

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To Prohibit Smoking in Federal Buil...

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BEFORE THE

SUBCOMMITTEE ON
PUBLIC BUILDINGS AND GROUNDS

OF THE

COMMITTEE ON
PUBLIC WORKS AND TRANSPORTATION
HOUSE OF REPRESENTATIVES

ONE HUNDRED THIRD CONGRESS

FIRST SESSION

ON

H.R. 881

TO PROHIBIT SMOKING IN FEDERAL BUILDINGS

MARCH 11; APRIL 22, 1993

Printed for the use of the
Committee on Public Works and Transportation

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103D CONGRESS
1ST SESSION

H. R. 881

To prohibit smoking in Federal buildings.

IN THE HOUSE OF REPRESENTATIVES

FEBRUARY 16, 1993

Mr. TRAFICANT introduced the following bill; which was referred to the Committee on Public Works and Transportation

---

A BILL

To prohibit smoking in Federal buildings.

Be it enacted by the Senate and House of Representatives of the United States of America in Congress assembled,

SECTION 1. SHORT TITLE.

This Act may be cited as the "Ban on Smoking in Federal Buildings Act".

SEC. 2. FINDINGS.

Congress finds that—

(1) environmental tobacco smoke is a cause of lung cancer in healthy nonsmokers and is responsible for acute and chronic respiratory problems and other health impacts among sensitive populations;

(2) environmental tobacco smoke comes from secondhand smoke exhaled by smokers and sidestream smoke emitted from the burning of cigarettes, cigars, and pipes;

(3) citizens of the United States spend up to 90 percent of a day indoors and, consequently, there is a significant potential for exposure to environmental tobacco smoke from indoor air;

(4) exposure to environmental tobacco smoke occurs in public buildings and other indoor facilities; and

(5) the health risks posed by environmental tobacco smoke exceed the risks posed by many environmental pollutants regulated by the Environmental Protection Agency.

SEC. 3. SMOKING PROHIBITION IN FEDERAL BUILDINGS.

(a) IN GENERAL.—On and after the 180th day after the date of the enactment of this Act, smoking shall be prohibited in any indoor portion of a Federal building and in any other space owned or leased for use by a Federal agency.

(b) EXECUTIVE BRANCH BUILDINGS.—The Administrator of General Services shall issue regulations, and take such other actions as may be necessary, to institute and enforce the prohibition contained in subsection (a) as such

prohibition applies to Federal buildings and other space owned or leased for use by an Executive agency.

(c) JUDICIAL BRANCH BUILDINGS.—The Director of the Administrative Office of the United States Courts shall take such actions as may be necessary to institute and enforce the prohibition contained in subsection (a) as such prohibition applies to Federal buildings and other space owned or leased for use by an establishment in the judicial branch of the Government.

(d) LEGISLATIVE BRANCH BUILDINGS.—

(1) HOUSE OF REPRESENTATIVES.—The House Office Building Commission shall take such actions as may be necessary to institute and enforce the prohibition contained in subsection (a) as such prohibition applies to Federal buildings and other space owned or leased for use by the House of Representatives.

(2) SENATE.—The Committee on Rules and Administration of the Senate shall take such actions as may be necessary to institute and enforce the prohibition contained in subsection (a) as such prohibition applies to Federal buildings and other space owned or leased for use by the Senate.

(3) OTHER ESTABLISHMENTS.—The Architect of the Capitol shall take such actions as may be nec-

essary to institute and enforce the prohibition contained in subsection (a) as such prohibition applies to Federal buildings and other space owned or leased for use by an establishment in the legislative branch of the Government (other than the House of Representatives and the Senate).

SEC. 4. REPORT BY ADMINISTRATOR OF GENERAL SERVICES.

Not later than 2 years after the date of the enactment of this Act, the Administrator of General Services shall transmit to the Committee on Public Works and Transportation of the House of Representatives and the Committee on Environment and Public Works of the Senate a report that includes—

(1) information concerning the degree of compliance with this Act; and

(2) an assessment of the legal status of smoking in public places.

SEC. 5. PREEMPTION.

Nothing in this Act is intended to preempt any provision of law of a State or political subdivision of a State that is more restrictive than a provision of this Act.

SEC. 6. DEFINITIONS.

For the purposes of this Act, the following definitions apply:

(1) EXECUTIVE AGENCY.—The term "Executive agency" has the same meaning such term has under section 105 of title 5, United States Code.

(2) FEDERAL AGENCY.—The term "Federal agency" means any Executive agency or any establishments in the legislative or judicial branches of the Government.

(3) FEDERAL BUILDING.—The term "Federal building" means any building or other structure owned or leased for use by a Federal agency, except that the term shall not include any area of a building that is used primarily as living quarters.

TO PROHIBIT SMOKING IN FEDERAL BUILDINGS

THURSDAY, MARCH 11, 1993

HOUSE OF REPRESENTATIVES,
SUBCOMMITTEE ON PUBLIC BUILDINGS AND GROUNDS,
COMMITTEE ON PUBLIC WORKS AND TRANSPORTATION,
Washington, DC.

The subcommittee met, pursuant to call, at 10:00 a.m., in Room 2167, Rayburn House Office Building, Hon. James A. Traficant [chairman of the subcommittee] presiding.

Mr. TRAFICANT. The subcommittee will begin its hearing on H.R. 881, a bill that bans smoking in Federal buildings.

Common perception of Congress is that lawmakers sit by the window, look out the window, assess carefully in which direction the crowd is moving, and then they go outside and get in front of the leading crowd. That will not be the issue here. This will not be a walk in the park.

The liability questions and the new information that comes out compels our government to look at this issue more seriously and critically than we have ever in our history.

With that, we will begin today to officially assess the information, scientific and factual data that has been prepared and created and submitted. We will deviate from our normal duties here today and our normal methodology by trying to accommodate one of America's new leaders who has to catch an airplane flight here about 10:20. So I hope that the Members of the Congress who were scheduled to appear first would not be offended and I thank them for accommodating us.

Before we continue, I would like to place into the record at this point a statement received from our Colleague from West Virginia, Congressman Nick Rahall.

[Mr. Rahall's prepared statement follows:]

STATEMENT OF REPRESENTATIVE NICK RAHALL

Mr. Chairman, I appreciate the opportunity to address the members of this subcommittee during your consideration of H.R. 881, legislation to ban smoking in all facilities owned or leased by the federal government. I have a number of concerns about this proposal that I respectfully urge my colleagues to consider as you evaluate whether legislation of this kind is necessary and whether it can be expected to have a productive impact on the federal workplace.

Mr. Chairman, let me describe my concerns. First, since 1986, when the General Services Administration issued guidelines on smoking in GSA-owned or -leased facilities, virtually all Executive departments and agencies have adopted policies to manage workplace smoking. Under the current restrictions, smoking in most federal facilities has been limited to specifically designated areas and, in the event of a con-

flict between smokers and nonsmokers, the wishes of nonsmokers prevail. There simply is no good evidence that this approach has failed.

A second problem with this proposal is that it ignores a number of important precepts which governed development of the original GSA guidelines. The first of these was the desire to accommodate workers who choose to smoke, as noted in the preface to the GSA rule, which states: "The proposed regulations also recognized the needs of those who smoke and permitted the designation of smoking areas. These areas were proposed to allow those who smoke to continue to do so conveniently and without affecting productivity. Hence, the policy attempted to recognize the rights, needs and concerns of all employees." This legislation completely abandons that important principle.

Third, this legislation also completely abandons the notion that individual departments and agencies of the federal government should have the flexibility to set the smoking policies that suit their workforce. Under this proposal, the same regulation would apply to the Department of Health and Human Services and to every small field office in the Department of Agriculture. It ignores the fact that employees in different offices may have very different preferences, that smoking bans may impose disparate hardships in different locations, and that conditions of occupancy and ventilation are not always the same. Individual departments and agencies are very well capable of managing smoking among their workforce, they have done so successfully to date, and they should continue to have the discretion to do so.

Fourth, this proposal fails to consider the rights of organized labor and the workers it represents, who consider workplace smoking a condition of work negotiable through collective bargaining. An AFL–CIO policy statement on workplace smoking notes that "unions have a legal responsibility to represent the interests of all their members—smokers and nonsmokers," and recommends that issues related to workplace smoking be worked out "voluntarily . . . in a manner that protects the interests and rights of all workers."

In fact, most of the unions whose members are directly affected by this legislation have expressed no dissatisfaction to me with current policies on smoking in the workplace, and are not likely to embrace more onerous burdens on a significant number of their members. All of these unions have more pressing concerns, chief among them how to keep their jobs. Many also are concerned about the overall problem of indoor air quality, which causes significant illnesses among many of their members.

In a recent survey of Social Security Administration workers, for example—many of whom are housed in General Services Administration buildings where smoking is banned or severely restricted—virtually all respondents who reported health problems on the job blamed poor workplace air quality for their illnesses. GSA is happy to support a ban on smoking yet has done little to address this more substantive concern, despite its public commitment to do so at the time it announced its regulation of smoking.

Mr. Chairman, I am aware that one of the primary motivations of legislation to ban smoking in the workplace is the risk assessment of environmental tobacco smoke released by the Environmental Protection Agency earlier this year. I am not a scientist, so I will not presume today to tell you that the EPA's analysis is unsupported. It is my understanding, however, that the scientific basis for this report is not unquestioned within the scientific community. In a number of recent news reports that have come to my attention, scientists from across the country have expressed concerns about the quality of the ETS science and the validity of EPA's conclusions. In one recent report, a well known epidemiologist quotes as follows a colleague he identifies as "an authoritative leader in the world of public health epidemiology": "Yes, it's rotten science, but it's in a worthy cause. It will help us to get rid of cigarettes and become a smoke-free society." The members of this subcommittee will hear testimony today from scientific experts that the EPA risk assessment of ETS is "rotten science," motivated more by a desire to end smoking than by the principles of objective research.

You also will hear testimony that whatever the merits of the EPA report, the primary question with respect to this proposal—what health effects may be expected from ETS exposure in the workplace—has not been answered. According to scientific experts, the EPA's analysis is a study solely of smoking in the home, and does not evaluate or make claims about the health effects of exposure in the workplace. It is not credible to suggest that ETS levels under current policies governing smoking in the federal workplace pose a health hazard to nonsmoking employees.

Nor does EPA have expertise in the area of the health effects of exposure to substances in the workplace. The Occupational Safety and Health Administration, which regulates workplace exposures, has evaluated this issue in the past, and has concluded that the data on workplace exposure to ETS are insufficient to determine

that a hazard exists. OSHA currently is re-evaluating the data on workplace exposure to ETS to determine if all of the evidence, including the EPA report, suggests that workplace smoking—and, in fact, all of indoor air quality—should be regulated.

Legislation based on alarmist reaction to the EPA report simply is not warranted. Neither should this subcommittee be swayed by arguments that smoking bans are good for smokers, since they may be encouraged to stop smoking altogether rather than endure the hardships of a total ban. It is not an appropriate function of the federal government to make lifestyle choices for its workers whenever the government, in its wisdom, considers those choices ill-advised. We may wish Americans would not smoke, but we cannot and should not make that decision for them.

Mr. Chairman, just last year, following implementation of a total smoking ban in Veterans Administration hospitals, the House of Representatives voted to require the Agency to set aside areas for smokers. In doing so, the House recognized that those who have served their country should not be subjected to unnecessary and arbitrary hardship, and that the preferences of all should be accommodated.

I will close today by encouraging my colleagues to show the same respect for federal workers. It is possible to accommodate the preferences of all employees without placing undue burdens on smokers or exposing nonsmokers to tobacco smoke involuntarily—the existing rules accomplish just that. We should not impose a change that accomplishes so little more at the expense of a significant portion of the federal workplace.

I thank the Chairman for the opportunity to address this important issue.

Mr. TRAFICANT. I would like to begin by introducing Dr. Antonia Novello, the Surgeon General of our Public Health Service. Doctor, welcome, and the time is yours.

TESTIMONY OF HON. ANTONIA C. NOVELLO, SURGEON GENERAL, PUBLIC HEALTH SERVICES, DEPARTMENT OF HEALTH AND HUMAN SERVICES, ACCOMPANIED BY DR. MICHAEL ERIKSON, OFFICE OF SMOKING AND HEALTH, CENTER FOR DISEASE CONTROL AND PREVENTION

Dr. NOVELLO. Thank you very much, Mr. Chairman. I thank you for inviting me to testify before the Subcommittee on Public Buildings and Grounds on the health effects of tobacco use and exposure to environmental tobacco smoke. The issue of environmental tobacco smoke is one of the most important, the most sensitive, and far-reaching workplace health issues of our time, and I am extremely pleased to be here this morning to assist you in your review of this important public health matter.

From a public health perspective, the issue is quite simple. Tobacco use and exposure to tobacco smoke are harmful and can lead to disease, disability, and even death.

Tobacco use is the most preventable cause of disease and premature death in this country. It kills more than 434,000 Americans every year, and is responsible for at least $65 billion in smoking-attributable health and economic costs every year.

On the average, smokers miss eight more days of work a year than nonsmokers. And every employee who smokes costs his or her company on the average at least $1,000 a year because of decreased productivity and increased health care costs.

Although this message is clear and succinct, we must constantly remind ourselves that what we are talking about is the premature deaths of nearly one-half million American people every year. And I think the best way to see it is this way: 434,000 people every year is the equivalent of three fully-loaded jumbo jets crashing every day and killing everyone, including the crew. So when you are looking at this many, smokers today are not the only ones today who

are at danger from tobacco smoke, but people exposed to the tobacco smoke of smokers are also at risk as well.

Environmental tobacco smoke, or ETS as we know it, is the combination of smoke exhaled by smokers and the smoke given off by the burning end of the cigarettes of smokers. Exposure to environmental tobacco smoke often is referred to as "passive smoking" or "secondhand smoking." But this ETS can cause disease, including lung cancer in nonsmokers.

In 1986, the Surgeon General of the United States concluded that the simple separation of smokers and nonsmokers within the same air space may reduce, but does not eliminate, nonsmokers' exposure to the tobacco smoke of smokers.

This was supported by a later recommendation by the Centers for Disease Control and Prevention's National Institute for Occupational Safety and Health that workers' exposure to ETS is most efficiently and completely controlled by simply eliminating smoking from the workplace. And most recently, the Environmental Protection Agency classified for the first time ETS as a known Group A human carcinogen.

I was invited here to explain the health effects of smoking and of being exposed to this secondhand smoke. Because of the role of this subcommittee in the Federal workplace, adult workers are a primary area of interest. And because Federal facilities serve and are open to children, and because of the issues surrounding tobacco use are far-reaching, I would be remiss if I did not at least touch upon the other critical concerns facing America when it talks about tobacco use.

As a Surgeon General, I have also devoted a large portion of my agenda to opening the eyes of the people who may not see the real hazards and risks of tobacco use. But it disturbs me tremendously that, as a Surgeon General and a pediatrician, we have sanitized the public health threat of tobacco use in this country.

Because tobacco use among our youths is not associated with immediate deaths or violent crime, we continuously perceive it in this country as less menacing and less problem than alcohol abuse or the use of illegal drugs.

That is a big reason, I am sure, why we utterly fail to enforce minors' access laws on tobacco or to restrict tobacco advertising and promotions that clearly appeal to youth, because we, just in this country, don't take seriously the problem of use of tobacco in our youth.

But let me remind you that each single day, 3,000 young people start smoking in this country and each year more than 3 million American children under the age of 18 consume 947 million packs of cigarettes, and that 10 percent of your fourth graders is already a smoker. So when I tell you that where there is smoke, there is children, I am actually being very serious.

Of the 3,000 young people who will become smokers today, about 30 will be murdered, about 60 will die in a traffic accident. However, 750 of them will die secondary to smoking related diseases during their lives. The consequences of tobacco use are real, and the targeting of the children by the tobacco industry is real, and we must put real enforcement teeth into the law to keep tobacco out of the hands of the children and the teenagers, and we must

take real steps to place limits on image-based advertising that preys on the insecurities of the youths of America.

Early last year, I joined with my colleagues of the American Medical Association in calling for R.J. Reynolds Tobacco Company to voluntarily withdraw its Joe Camel Advertising Campaign. This is a campaign that shamelessly appeals to the children through the use of what has been shown to be one of the most widely recognized cartoon characters among young children today.

I wonder if it would shock you to realize that 30 percent of three-year-olds, and I can tell you too that more children than any other utilization scheme has recognized Old Joe Camel than Mickey Mouse when we are talking of cigarette advertisement. And since that infamous campaign, the number of young people that smokes the brand of cigarettes that Old Joe pushes has risen to 25 percent of those who happen to be underage smokers.

It is up to us, the leaders, to help put a stop to the tobacco industry's seduction of our children.

Easy access and aggressive marketing have also been responsible for the alarming upturn in the use of smokeless tobacco under the rubric that it is less important or has less complications. Or more accurately, I would like to call it spit tobacco, not smokeless tobacco, because, among our Nation's youth, they believe it might be less harmful because it has no smoke. One out of every five kids in 22 States in this country uses it today. And the average age is nine and some of them are starting as early as five.

So last year, I released a report showing the use of spit tobacco by our young people recently and rapidly climbing to very high levels. With youngsters using moist snuff tripling in five years, I am extremely worried.

My cause for alarm is the fact that spit tobacco is just as addictive as cigarettes because, after all, nicotine is nicotine, and tobacco is tobacco, whether you smoke it, whether you chew it, or whether you spit it.

And it seems to me that when you see the epidemic of lung cancer following widespread cigarette smoking in this country, I fear if we do not pay attention to the spit tobacco that is being used by youth today, this country is going to see an epidemic of oral cancer in the decades ahead. Smokeless tobacco has the same risks as the tobacco that you smoke.

Children are not the only targets of the tobacco industry. As a Hispanic woman, I am especially concerned about the seduction of women and minorities by the tobacco industry. To the average American, the slogan "You've Come a Long Way, Baby" might just be another cute advertising ploy, but to those concerned about the dangers of tobacco, the meaning is more serious and goes much more deeper than that.

Women and minorities in this country are continuously being exposed to numerous advertising messages that subtly encourage them to begin a lifelong, often deadly addiction to smoking; an addiction that was not present in this country in women prior to the 1900s.

As a result of the widespread, seductive courting by the tobacco industry, lung cancer deaths in women have outnumbered the deaths caused by breast cancer. If smoking trends continue, women

may eventually equal men in the prevalence of smoking. And it is a real harsh reality that women that smoke, like men, are absolutely going to be predestined to die like men.

It is up to us leaders to see that the Virginia Slims Woman does not follow in the tragic steps of the Marlboro Man, but instead follows a different path that will lead to greater health and fulfillment.

Mr. Chairman, members of the subcommittee, let there be no doubt that the health effects of smoking and passive smoking are real and preventable, which means that something can be done to eliminate them. The scientific case against environmental tobacco smoke justifies—and should compel—action to protect people, specifically children, from the potential health effects of exposure to environmental tobacco smoke.

To reduce the health risks, restrictions to prevent exposure must be more comprehensive than those currently in place in most facilities. Studies show that 75 percent of our citizens do not smoke. They should not have to endure the consequences of the smoke of others. We must help them claim their rights to clean air. In the quest of protecting the rights of smokers, let's not forget to protect the rights of the nonsmokers as well.

In closing, let me restate that the general direction of H.R. 881 would provide important public health safeguards for those who work in and visit Federal buildings. The Department of Health and Human Services supports the objectives of H.R. 881.

By dealing with this health issue, this subcommittee has assumed an important leadership role in public health. I commend your consideration of this important issue.

This concludes my formal statement. I would be more than happy to answer any questions that you or any other member of the subcommittee might have. And I would like to request permission to bring Dr. Michael Erikson, the Director of the Office of Smoking and Health from the Centers for Disease Control and Prevention to answer any technical questions that might arise.

Thank you, Mr. Chairman.

Mr. TRAFICANT. Granted. Mr. Erikson, if you would like to come forward.

It is an unusual start for us, for the Members to accommodate this schedule of our Surgeon General. We appreciate your frankness, your comments, and your willingness to speak out and not mince or choose words.

I think your statistics of 434,000 Americans each year that die due to smoking, one out of every five deaths, statistics of $65 billion a year, and there are some that are even higher, Doctor, that each and every smoking employee costs their company at least an average of a thousand dollars a year in increased health care costs, these statistics have been widely circulated and spread.

How did your office gather those statistics and how did you further verify those statistics?

Dr. NOVELLO. Well, I can tell, when you are the Surgeon General, I don't have too much difficulty gathering statistics. And one of the issues is that the people do constantly make us aware of what is out there, specifically all the coalitions out there that deal with smoking. But perhaps I would yield to doctor Michael Erikson,

if there is any more recent data that supports the $65 billion in economic costs to society.

A very well-known number that the Office of Technology Assessment estimates, that current smoking-related health care costs range from an estimate of 11 billion to a high estimate of 35 billion, with an average of 22, which really means that this representation—3 to 9 percent of total U.S. health care spending with a median estimate of 22, putting it close to at least 6 percent of all United States health care spending just related to smoking.

Mr. TRAFICANT. Doctor?

Mr. ERIKSON. There are a number of data sources for the statistic that you quoted. Most of them are published in the Surgeon General's report that comes out annually. The first relevant report on this topic was the 1986 Surgeon General's Report on Involuntary Smoking which concluded that involuntary smoking causes lung cancer in healthy adults and that mere separation of smoking into separate sections isn't adequate.

Subsequently, a number of other reports which came out most recently, and I know you will hear this morning from the EPA on their risk assessment on the health effects of the environmental tobacco smoke, was released in January and they will be answering specific questions.

As Dr. Novello also said, the economic estimates come from the government itself from the Office of Technology Assessment in terms of the health care expenses and the overall costs of smoking.

Mr. TRAFICANT. Your office released a report that concluded that ETS can cause lung cancer in nonsmokers. What was the public reaction to that report, if you could, and has your office completed any further reports on that issue since then?

Dr. NOVELLO. Well, I can tell you that, from the first report of the Surgeon General in 1986 really tying ETS to lung cancer, we have gotten, I would assume, the same kind of calls you all are getting by which there will always be people pro and people against. But at the same time, the National Institutes of Health was doing coordinating research on the same subject and they have reiterated the findings of the EPA.

At this particular time, the EPA report which came forward in January of this past year, and the 1986 Surgeon General's Report have already proven the case for passive inhalation being as dangerous as any other kind of form of smoking.

And maybe Dr. Erikson might be able to add much more to this.

Mr. ERIKSON. Just in terms of the impact of the report that came out in 1986, the Surgeon General's record, at that time, approximately 27 percent of private sector workplaces restricted smoking in the workplace at the time of the first Surgeon General's report on involuntary smoking, 27 percent.

Today, 1991 figures indicate 85 percent severely restrict or prohibit smoking in the workplace. That is the type of change that has occurred as a result of the provision of information documenting the problem.

In terms of smoke-free workplaces, in 1987, we estimate 7 percent of companies were smoke-free. And today that estimate is closer to a third of private sector workplaces that are smoke-free. And that movement in the private sector is a direct result of the in-

creased information we have on the health effects of passive smoking.

Mr. TRAFICANT. Have you performed any studies or is there any raw data that now begins to show the development of a new disease factor in workmen's compensation cases or workers in the workplace that may have been subjected to smoking, secondhand smoke, as claiming dysfunction and/or impairment or reduced capability?

Have there been any of those cases that have started to surface? One of the concerns of our committee certainly will be that this is now a new ball game. This is not just a smokers rights issue, and a health issue. There is a tremendous exposure by Uncle Sam that perhaps there will be a new wave of medical litigation and claims for dysfunction.

Have you performed any studies in that regard, or do you have or are you preparing any collateral or individual studies to assess what that impact could be on our government and the operation of our government?

Mr. ERIKSON. Everyone is expecting there to be an increase in case law regarding this issue of nonsmokers seeking relief through regulatory or legal channels through exposure in the workplace. In fact, some cases have already been introduced in the courts and are being deliberated as we speak.

From an observational standpoint, when companies go smoke-free, the lead in companies going smoke-free often does not come from the Medical Department but from the Chief Financial Officer of the company or the Risk Manager of the company who is concerned about these very same liability issues.

Our sense is that that sense of concern of liability has nothing but increased, given the release of the EPA record which now classifies ETS in the same way as other occupational hazards such as asbestos and benzene, while we are very much in the stage of government of responding to this. But there is very much a concern that liability will increase for employers.

Mr. TRAFICANT. It would be safe, then, for us to assume that, since the 1986 report of your office and now an additional report by the Environmental Protection Agency, that there would be an even more compelling legal position to document that our Federal Government has, from within its own structure, identified a matter that provides such liability for the future. Would you agree to that?

Dr. NOVELLO. What I would like to say, Mr. Chairman, is that the American public is very much keen on knowledge, much more than when we were growing up, and more importantly, with the reports that came out with findings that really show the damage for children, equally to adults.

In the presence of that data, I have the feeling from the public opinion calls that my officers are getting, because if the government has put this out in January 7 of 1993 with the SG Report of 1986, that means that this is real.

Knowing that our buildings are visited not only by adults but by children, and considering that the first reports only really testify to the fact of lung cancer and the issue pertaining to adults and the new findings now of the bronchitis and the pneumonia and the kids with the upper respiratory infections and the worsening of

asthma and the aggravation of ones who suffer it, it seems to me at this stage of game, that people are clamoring for what it is with facts that are already documented by the government of the United States. And from that sense, many more things will come down.

But having been reported after having been two years in the making, people were looking forward to responses from us and responses for all the people involved in this. But public opinion out there is mounting by which they are really looking forward to see that some findings, which places it as a human carcinogen for the first time, are looked upon closely to prevent disease and disability in the face of the factual data.

That, before, was not obtainable with the exception of when you smoke, babies of mothers who smoke will be less weight and, therefore, complications in the long term. Now, once they are born, there are complications that mothers and fathers can see and, therefore, they would want to be able to protect them now when the information is available.

Mr. TRAFICANT. Dr. Novello, I would ask that the 1986 report of your office be submitted. I ask the members' of the subcommittee unanimous consent to have that 1986 report of the Surgeon General be received for the record, and be placed in the subcommittee files.

Hearing no objection, so ordered.

[The Report of the Surgeon General, "The Health Consequences of Involuntary Smoking" (1986) was received for the subcommittee files.]

Mr. TRAFICANT. Before I move to our Ranking Minority Member, I ask unanimous consent that the following communication from the White House be placed in the record. To Representative Traficant and I quote, "I strongly agree that discouraging harmful behavior such as smoking is an essential part of our overall effort to improve the health of our citizens. For this reason, I sincerely regret that scheduling conflicts do not permit me to join you on Thursday.

I do hope that we will have many opportunities in the future to work together on this issue." From the First Lady, Hillary Clinton.

I ask that that be placed in the record. I ask unanimous consent that that be placed in the record. Without objection, so ordered.

[The White House communication follows:]

THE WHITE HOUSE

March 9, 1993

Dear Representative Traficant:

Thank you for inviting me to testify at your Subcommittee hearing on the effects and costs of smoking on America's health care system.

I strongly agree that discouraging harmful behavior such as smoking is an essential part of our overall effort to improve the health of our citizens. For this reason, I sincerely regret that scheduling conflicts do not permit me to join you on Thursday.

I do hope we will have many opportunities in the future to work together on this important issue.

Sincerely,

Hillary

The Honorable James A. Traficant, Jr.
U.S. House of Representatives
Washington, D.C. 20515

Mr. TRAFICANT. I would like now to recognize the distinguished Ranking Minority Member from Tennessee, Mr. Duncan.

Mr. DUNCAN. Thank you, Mr. Chairman.

Dr. Novello, thank you for coming here this morning and for your statement that you have given. As I mentioned a few days ago at a hearing on this bill, I am a nonsmoker and grew up in a family where nobody smoked. And I speak in many, many schools now and I always—frequently, I try to encourage the children and the young people not to start smoking. And you devoted much attention to that in your statement.

And I have read that many, many people who smoke wish that they didn't, a very high percentage. I know that almost all people who smoke, I am sure, would encourage their children not to do so.

And yet saying all that, what I am wondering about is this: And about one-fourth of our population or maybe over one-fourth, I don't know, 50 or 60 million people, I suppose, smoke. We all know many fine and wonderful people who smoke.

Is there a way—I mentioned this a few days ago, but we didn't really get into it. Is there a way to be anti-smoking without being anti-smoker, and a way to balance the rights of the smokers and the nonsmokers?

Dr. NOVELLO. Well, Mr. Duncan, your question is a wonderful one, because I really think the important issue here is we are anti-smoking not anti-smokers.

I remember when the Surgeon General report of 1990 came out, and it was suggested that it is never too late to quit. In the quest of looking into the smoking in the buildings, it seems to me, if someone would like to quit as a consequence of this new information coming into the workplace, they will be welcome to do so. But if they do not want to, it should be their choice.

The issue is when and if they want to quit, that they realize when you decide to quit before the age of 50, even though you have been a long-term smoker, your life gets seven years added, and they should know about positive things that happen when you quit along with the negative things, which I think we have been telling people all along.

But if we look at this in any close way in which we want to help the smoker, programs will have to be in place and options for the smokers to receive programs, quitting techniques and the like, together with counseling need to be available.

But whatever we do in the future, it seems to me that there are 50 million people in this country who still smoke. And 50 million people that should not be able to go underground when what we are trying to do is make sure that the rights of the smokers that have always been respected or equally respected as the rights of the nonsmokers. A fine median will have to come, but the time has come when both rights are respected.

Mr. DUNCAN. In some of the press reports that came out about the EPA report on the secondhand smoke, the 3,000 deaths, usually some place in those stories there were some suggestions or charges or accusations that the science on which that report was based was in some way flawed or not really accurate.

Have you studied that report or looked into the science behind it? Do you think that report was in any way exaggerated or flawed, or do you have any questions in your own mind about that?

Dr. NOVELLO. Well, I can tell you one thing. One thing that I find as a scientist and a physician is that, for every report and for every article that comes that finds something good, there is always someone that is going to find something wrong. It all depends on what you want to believe. But from the EPA report as a whole, I can tell you that it has a solid basis of science data, and more importantly which I think brings the point home, the chairman of the Scientific Advisory Board of the EPA report who has received research funding from the Council on Tobacco and Research was part of the group that voted unanimously to say that the science involved in the EPA was accurate.

So when you have that kind of input, you realize that the data there was not flawed, in spite of the fact of all the comments that we find with the people who would like to make it so.

Mr. DUNCAN. The Surgeon General's Office started the warnings about smoking. What do you feel has been the effect? It seems to me that there is a real anti-smoking feeling out all across the country, but has the percentage declined as much as you thought it would have based on all that's gone into it?

Dr. NOVELLO. Well, when we talk about the issues of labels, I would like to talk about it as a researcher and as a pediatrician. I think we are going to need much more research on labels. Have we found a diminution in the smoking because of the label reading "may cause cancer"? Or would we find more diminution of smoking if it says "does cause cancer"?

Have we found any diminution in smoking if the label says "it may complicate pregnancy" or would we find a more drastic diminution if it says "it does complicate pregnancy"? I have a feeling that we will have to have more research into the labeling issue as it pertains to the people of today with 10 years accumulative data of what has happened in the tobacco field and specifically in the persons of the case where the warning of the Surgeon General is meant to be a public health warning for people to avoid risk so more research, absolutely, is going to be found.

What I find out there is that the youth in itself, even though it is being not going very much hype, the women within the youth seems to be also smoking equally as men. And more importantly and very importantly, the African-American youth is the one who basically has dropped the smoking trend.

But more research we are absolutely going to need into the issues of the label.

Mr. DUNCAN. I guess some of what I am wondering about, I think people pay attention to some of the stories that come out like on the EPA report and such statements that you have made and efforts by others. But I am not so sure that anybody much pays attention to the warnings that are on the cigarettes anymore. You know we have just sort of grown accustomed to those.

Let me ask you this: I have been told by people who smoke that they would like to quit. They would very much like to quit, but that it is—that it is an extremely difficult thing. And I am wondering,

are these patches working? Are these smoking cessation classes working? Is there anything out there that is working today?

Are we asking Federal employees to do something that is just almost going to be impossible for them to do. I know many, many people have quit, but there have been so many millions of others who have tried and failed.

Dr. NOVELLO. I think the important thing here, Mr. Duncan, is clarification of the issue. With this trend, we are not pushing people to quit. We just want people not to smoke in the workplace. There is a difference. If in doing so, the ones who would like to take the opportunity and stop smoking because it would be a little bit difficult for them to go outside to smoke would like to take the bandwagon and quit then therefore they have to realize that the nicotine patch is something that should assit in the desire to quit, aided by group therapy by which you really get reinforced by the benefits of what you have done.

But one thing does not go with the other. And I can tell you that I just came from speaking to the Blue Jays and the Phillies, by which I talked about—to the pitchers, how much I appreciate whenever they speak in front of the camera they did not become an advertisement for spit tobacco.

One of the things that came out of there is absolutely you must want to quit, and if doing so we are helping in particular the baseball teams with quitting programs, with counseling programs paid by the National Cancer Institute and with a nicotine patch offered for free, so when the players want to quit, there will be ways by which we will help.

But doing in—provision quitting is not the friend that we want. But by making sure you are in the working places, there are no smoking in the place as a whole, and not necessarily quitting by the people who still want to say, considering that it takes about seven tries on anyone to really quit.

Mr. DUNCAN. Let me just ask you very quickly, because we need to move on to other Members. But you mentioned some study or report that said that the average age of starting with a chewing tobacco was nine and that one in five children are doing that. Where did that come from?

Dr. NOVELLO. I requested a report from the office of the Inspector General from HHS and it just came out six or seven months ago. And it was particularly the day of the first storm in Washington, so there was very little media regarding that. I could submit it for the record with all my comments in it, but at this stage of game, regardless, in these United States, one out of every five white males uses spit tobacco.

It is a problem not of just rural—but of all America, but white males in particular in 22 states and territories of the United States that chew tobacco for four different reasons. One, parents believe that it is cheaper than candy and therefore why should they worry and at least they do not have smoke bothering anyone.

Second, it is the most common free sample given of the tobacco products out there today.

Three, there is a terrific tie to baseball and kids believe when they grow up, they want to be like these gorgeous players, and

more importantly, there is not enforcement of the law. And one-third of parents of chewers have chewed tobacco themselves.

In the presence of that, we have an epidemic of oral cancer in this country in the making that no one is paying attention under the rubric that smokeless is less harmful. That's why I said tobacco is tobacco, whether you smoke or chew it or spit it.

In that sense, I would be more than glad to submit that report for the record because you would be surprised that the mean age of beginning is nine and the average is five. On Native American reservations, it is even 91 percent of children in seventh grade in North Dakota of one tribe already spits tobacco.

Mr. DUNCAN. I would like to ask for unanimous consent that Dr. Novello be allowed to submit that report. And also that any other witnesses that we have today be allowed to submit any comments or reports that they wish to that they may not have brought here today on liability issues or any type of issues pertaining to this hearing.

Mr. TRAFICANT. Without objection, so ordered.

[The Report, "Spit Tobacco and Youth," was received and may be found beginning on page 220.]

Mr. TRAFICANT. We now go to the distinguished gentlewoman from the District Eleanor Holmes Norton.

Ms. NORTON. Thank you, Mr. Chairman. Before I ask my questions, I ask unanimous consent my opening statement be admitted to the record.

Mr. TRAFICANT. Without objection, so ordered.

[Ms. Norton's prepared statement follows:]

STATEMENT OF CONGRESSWOMAN ELEANOR HOLMES NORTON

Chairman Traficant, I comment you for your interest in curbing the dangerous effects of secondhand smoke and am pleased to join my colleagues today to consider H.R. 881, a bill to prohibit smoking in Federally owned and leased buildings. I extend a special welcome to the distinguished Members of Congress and other witnesses who are here this morning to share valuable information with us.

Tobacco smoking has long been recognized as a leading cause of disease and death. In more recent years, medical and environmental assessments have revealed the hazards of secondhand smoke. There are strong indications that non-smokers who are exposed to smoke exhaled by smokers or given off by burning cigarettes may also be at risk for developing some of the negative health effects associated with smoking.

Greater awareness of the risks of passive smoking has led to policies that protect non-smokers. For example, airplane passengers cannot smoke during domestic flights. Smoking on buses has long been prohibited. In some cities, such as New York, there is already a total ban on smoking in municipal government buildings. And increasingly, private companies throughout the country have completely barred smoking in their facilities.

By prohibiting all smoking in federally owned and leased buildings, H.R. 881 likewise brings federal smoking policies in conformity with health and scientific findings. While some may argue that designating separate smoking areas is a more reasonable measure, the protection of non-smokers is only possible when such smoking areas are individually ventilated to avoid the recirculation of secondhand smoke. The expense of installing separate ventilation systems in existing federal buildings would be unfeasibly high. H.R. 881 is a cost-efficient measure with a most admirable goal. This bill will protect federal employees from the hazards of breathing secondhand smoke and provide them with a healthy environment as they continue to serve the American people. Again, my thanks to Chairman Traficant and to our witnesses here today.

Ms. NORTON. Dr. Novello, the government has done for years now a very good job in exposing the research indicating the harm-

fulness of cigarette smoke. One is left to wonder whether it has done as good a job in research to help with the addiction of smoking itself.

My colleague, Mr. Duncan, raises a question that I think is an important one, about the smoker who finds himself now surrounded by data and by sanctions and restrictions. Is the government—I am told that addiction to smoking can be and often is a more serious addiction, harder to break than addiction to heroin.

Is there in place research at NIH or elsewhere that could develop chemical or behavior aids so that smokers themselves would quit and this problem of secondary smoke would diminish more quickly than it is?

Dr. NOVELLO. I think that your point is excellent, Ms. Norton, in the sense that, as we said before, we are not anti-smokers, we are anti-smoking. And, therefore, the ones who do want to quit, we want to be able to be part of that help. The National Institutes of Health is doing particular research because we don't want 50 million Americans to go underground for something they cannot avoid doing.

Nicotine is very addictive. It was even shown in the Surgeon General's report that Nicotine is an addictive drug.

Ms. NORTON. Dr. Novello, I am concerned that smokers are left to people who come on television who say they can help. These people are not legitimate. I do not know of chemical or pharmaceutical aids, or at least any significant numbers of them. One would think that the country that leads the world in science might be able to develop aids for this addiction and I must say other addictions as well.

Any information that you would like to supply for the record on the state of current governmental research in this, helping to eliminate addiction to smoking, I am going to say other kinds of addiction as well, would be very useful.

Is there any information or data to show that requiring that there be a separate space in a building has any effect to reduce smoking itself?

Dr. NOVELLO. The Surgeon General report suggested in 1986 that the mere notion of dividing smokers and nonsmokers in one single space absolutely does not diminish the problem. Some other studies have suggested that perhaps different rooms with separate ventilation might do so, but it seems to me that the cost would be so prohibitive.

Ms. NORTON. No. No. My question is whether or not people stop smoking, whether there is any indication that people stop smoking when you tell them they can't smoke, they must go to a separate room to smoke.

Dr. NOVELLO. There has been data that shows that maybe because of the annoyance of having to go out and smoke rather than do it there, it makes you smoke less. It might not make you stop, but it makes you slow down. And even if you slow down, the risks would be probably not as much because of the less content of smoke that you inhale. But do you want to——

Mr. ERIKSON. There have been some studies that have been published in peer review literature on this and I would say the evidence is mixed right now about whether smokers will stop smoking

as a result of smoke-free workplaces. Some studies indicate they do. Others say they just switch where they smoke. They smoke off the job more to compensate and still smoke the same amount.

I think that the two points are that—one is that the major goal and objective of this type of work is to protect the nonsmoker and that it does, unequivocally. Secondly, we do think it creates the right message to smokers and we think it will help with preventing relapse for those smokers who are trying to quit and don't have to stay in a smoking environment. It is easier for them not to smoke again if they aren't exposed to it.

We have found in published research that smokers accommodate these types of restrictions, also, quite well. That there are some anticipations that will be a burden, but in reality, it is not a problem at all.

And I would just encourage you to talk to private sector companies that have gone smoke-free or even the Department of Health and Human Services, Humphrey Building, and all of their facilities being smoke-free and talk to the people. You will see that it is not a hardship and no one would want to go back to an environment where smoking is allowed. And we do think it is helpful for the smoker.

Ms. NORTON. Following that logic, if people go one step further outdoors, especially in the wintertime, does it follow that they might smoke even less or perhaps really be persuaded to quit?

Dr. NOVELLO. I can tell you that when I came into the Humphrey Building in 1990, one almost used to be clouded in smoke when one walked into the front door. And the more I walk through that door now, the less people I see smoking, even in the winter. So somewhere on the way, maybe the time you have to go down and smoke down there, will deter you. Don't do two an hour. You might do one every three.

And if that's the case, even subtly, we are letting people smoke less because they won't want to be bothered to go outside in the winter. Whatever helps might be needed, but we are not anti-smokers.

Ms. NORTON. Some considerable inconvenience. There looks to be some correlation between the degree of inconvenience and the amount of smoking at least that goes on.

Finally, are there any studies that when people are isolated in a room, disease on the part of the others in fact goes down? Do we know that yet?

Dr. NOVELLO. You mean that, for example, if we know that there is runny nose and aggravation of asthma and the like, when you separate the smokers from the nonsmokers with the aspiration of the smoke, does this go out?

Ms. NORTON. That, and cancer and bronchitis and emphysema and the rest of it.

Dr. NOVELLO. At this stage of the game, I don't know if we have enough follow-up on that since the EPA report came out January 7th. But from the short term, I can tell you that people say that they can breathe easier. Their wheezing is less and the runny noses have grown less.

But I also know of people who have, even in the smokers area, have gone for tests and have had nicotine found in their urine,

even though they have never smoked. So absolutely when they went back three days later, after this was totally banned, there was not nicotine in their urine.

So there is cause and effect to think that this will come in a better fashion once the people pick up that, separating them makes an effect in the other ones who get the consequences of smoking in their body.

Ms. NORTON. Thank you, Dr. Novello.

Mr. TRAFICANT. I know Dr. Novello has to leave shortly. I would like to ask each Member if they would ask their most important question and submit the rest of their questions in writing and we will have them answered for you.

I yield to the gentleman from Missouri, Mr. Emerson.

Mr. EMERSON. Thank you, Mr. Chairman. I understand we are going to hold a hearing beyond today's hearing on this subject and I wonder if it might be possible, given the time constraints of Dr. Novello today, if she might appear at some other time when we are holding a hearing on the subject?

Dr. NOVELLO. I would be more than glad to consider it.

Mr. EMERSON. Dr. Novello, you are a forceful advocate of promoting smoking and ETS as a public health issue. And at the same time, the government's general focus on the AIDS epidemic is directed at the civil rights of people who remain HIV+. Why the disparity in treatment of these two health issues and do smokers have any civil rights?

Dr. NOVELLO. Well, I believe absolutely that smokers have civil rights and I think I have been mentioning that for a while. Of the difference with AIDS, there is certain behavior that put you at risk and the difference with passive inhalation, you have done nothing to die of lung cancer except to be in the presence of someone who smokes. There is a difference right there.

Mr. EMERSON. Where you advocate abstinence is an appropriate approach to each public health issue but, of course, you have the means of enforcing abstinence in one instance but not in the other.

Dr. NOVELLO. But if you look at the data even abstinence advocation has not worked for 54 percent of the adolescents today and obviously abstinence of smoking has not worked for 50 million Americans today out of 249 million. The issues are smokers have rights and nonsmokers have rights.

Now, are we going to fit the pattern when we know for the first time that smoke is a carcinogen and be alert if we are going to prevent cancer in this country on the basis of data that we have today? How responsible should government be to be able to deal with this before the epidemic begins. I think then the rights of both have to be taken into consideration but the passive rights, inhalers' rights, have to be respected equally.

Mr. EMERSON. Is it a fact that the EPA document that is the focus of so much attention here is a controversial document, that there is a scientific there is not scientific unanimity on the points made by the EPA record; is that true?

Dr. NOVELLO. You are correct. The thing that happens is the same thing that happened in 1964 when Dr. Luther Terry came with the first report that tied lung cancer to tobacco. Controversy erupted then and now almost 30 years from that anniversary in

January of this coming 1994, we have come across much more things that were prompted by that controversy.

It seems to me we will see the same thing in the year 2010. The issue, when it was time to vote as a unison group on the report of the EPA, even the chairman of the executive board was able to vote in a unanimous consent that scientists that know what they are doing agree with the data as flawless—controversial—but flawless enough to be able to put into the front as a document that said carcinogen is smoke and that smoke is acting as a lung cancer producing agent and the government was able to say so in January 7th 1993.

Mr. EMERSON. Well, I do think we do need more science on the subject. Let me share with you, there was a news report that has been called to my attention entitled, "Is EPA Blowing Its Own Smoke," referring to the ETS risk assessment and raising a number of concerns about the scientific quality of the ETS risk assessment.

And there was quoted in the article one observer said it is now open season on whatever contaminant the EPA chooses to label the killer contaminant of the week with the effect that, once again, Americans are going to be stampeded into fearing a substance for reasons which, upon close inspection, are scientifically indefensible.

And a Yale professor, a Dr. Alvin Feinstein, was quoted in that same article that a prominent epidemiologist had recently stated that, "yes, it is rotten science, but it is in a worthy cause. It will help us to get rid of cigarettes and to become a smoke-free society."

It seems that the attitude of some at EPA is that there is no place for bad science except for a good cause. And we will decide for the American people what is a good cause. And I just want you to comment on that, if you would. I just want to assure ourselves that we are dealing absolutely with reliable people.

Mr. TRAFICANT. Would the gentleman yield before the doctor? I think it is a question that should be posed to the EPA, and I do not mean to stop your response. I hope we can move on to a couple more Members, too, if we could. If we could do that, I would like that question revisited by the EPA.

Mr. EMERSON. Well, I think the Surgeon General, seasonally works with the EPA and is relying somewhat on EPA data. And so I don't think it is out of order to ask that question of the Surgeon General.

Dr. NOVELLO. I can only tell you that the Surgeon General of the United States will not talk about any report which I do not trust. One right, EPA more importantly for every record that comes out and specifically comes out in the press rather than in a peer journal. I have doubt of the perception versus the reality.

And considering that the EPA was based on 30 case studies by very renowned scientists, I tend to go much more with a scientist when a scientist speaks rather than just the press.

Mr. EMERSON. Thank you, Mr. Chairman.

Mr. TRAFICANT. The Chair now recognizes the gentleman from South Carolina, Mr. Clyburn.

Mr. CLYBURN. Thank you, Mr. Chairman. Mr. Chairman, I request unanimous consent to have my opening statement entered into the record.

Mr. TRAFICANT. Without objection.

[Mr. Clyburn's prepared statement follows:]

STATEMENT OF HON. JAMES E. CLYBURN

Mr. Chairman, fellow members of this subcommittee, I thank you for allowing me to address some concerns I have about H.R. 881, a bill to ban smoking in all federally owned and leased office buildings. Banning smoking may not be as simple and uncomplicated as it appears. To begin with, for practical reasons, such a ban is likely to have extremely harsh effects on individuals forced to go outside when they wish to smoke. Treating federal workers in this manner is likely to undermine both morale and productivity.

In addition, this ban gives no consideration to other ameliorative measures, such as improved or separate ventilation in federal buildings which would improve indoor air quality in general.

The complete smoking ban proposed by this bill also would interfere with collective bargaining rights and would extend the discrimination that already exists against smokers in the federal work place. In a policy statement addressing work place smoking issues, employee representatives have stated, and I quote, "issues related to smoking on the job can best be worked out voluntarily in individual work places between labor and management in a manner that protects the interests and rights of all workers and not by legislative mandate." There is much merit to the employee's approach.

It stands in stark contrast to the position of the General Services Administration, one of whose representatives testified before this subcommittee recently in support of a smoking ban. He conceded that GSA had not surveyed affected workers to determine whether they believe such a measure is necessary. In addition, there is no need for Congress to consider smoking ban proposals at this time. Shortly before her departure from office, the former Secretary of Labor announced that she was directing the Occupational Safety and Health Administration to begin rulemaking concerning occupational exposure to environmental tobacco smoke. She also directed her staff to prepare a report for incoming Secretary Reich describing the issue and the rulemaking alternatives. OSHA meanwhile has received and evaluated numerous technical comments on indoor air quality issues.

There is no reason to believe that Secretary Reich and the Clinton Administration will not take appropriate and timely action on the ETS issue. Congress should give the Administration a chance to make decisions that are well within the competence and regulatory jurisdiction of the executive branch. We should not second-guess the Administration before it has even had a chance to consider this question.

I also note that with the announcement of the President's economic program, Congress has more than enough pressing national business to consider without taking on a proposal that is highly contentious and for which a need has not been demonstrated. There is enough pain in that package for federal employees as it is; we should hesitate to impose additional hardships and burdens.

I believe Congress should go about the important legislative business it has before it, and leave the question of work place smoking to OSHA, where it properly belongs.

Mr. CLYBURN. Thank you. Madam Surgeon General, I notice you indicated that in the Humphrey Building, I believe, you don't see the clouds now that you used to see. Do they have a designated smoking area in the Humphrey Building?

Dr. NOVELLO. Outside of the building.

Mr. CLYBURN. You go outside of the building. Have you made any attempts or have there been any studies made in order to determine what the inconvenience versus the loss in productivity, means in terms of overall performance of your employees?

Dr. NOVELLO. I don't think we have done that research yet, but I think many researches like the one you are proposing will be as a response of the EPA report. But at this stage of the game, it seems to me that I will take the inconvenience and the lack of productivity versus a cancer in my household any time.

Mr. CLYBURN. I would suspect that you would. I think we all would. As I said at a previous hearing, I lost both my parents to

cancer but neither one ever smoked, I don't know if that is a real connection.

Dr. NOVELLO. You are from South Carolina. I would watch passive inhalation.

Mr. CLYBURN. Well, I think that you would agree with me, then, that in the absence of some more scientific data and study on both these subjects, we would do well to adhere to what your premise was. You are not anti-smoker but anti-smoking.

Dr. NOVELLO. Yes, absolutely so, sir.

Mr. CLYBURN. No. Absence of scientific data to the contrary, maybe we ought to adhere to the 1986 study?

Dr. NOVELLO. I think what we are saying that we might not have a lot of scientific data to see what is the loss of productivity by the people who go out, but we do not have lack of scientific data that said smoke that causes cancer and passive smoke causes cancer, 3,000 a year. So I think that there is a clarification to make to the point.

Mr. CLYBURN. Well, my last question is the constraint of time. And, hopefully, we can get back to this when you return to us. If there is a building as there was in my instance before coming here, where there is a separate designated smoking area that is separately ventilated from the rest of the building, would that address itself sufficiently to the health questions that concern you, while at the same time granting the rights and privileges to smokers?

Dr. NOVELLO. It has been said that when you separate smokers from nonsmokers in one area, it makes no difference, you are going to passively absorbets anyway. It has also been said that when you want to ventilate separate rooms for smokers and nonsmokers, that could be a feasibility. The construction of such a place might be very much costly and then the location where this is implemented would be a decision.

Where would you like to spend the monies, either isolating them and make them more comfortable or helping them quit if they so desire and making them healthier? It should be the choice of the locality.

Mr. CLYBURN. In interests of time, maybe we will revisit this at a later time. Thank you.

Mr. TRAFICANT. The Chair now recognizes the Chairman of the Public Works Committee, Mr. Chairman, Norman Mineta.

The CHAIR. Thank you very much, Mr. Chairman. And I have no questions. I appreciate your testimony. I apologize for not being here earlier, but I want to thank you very, very much for not only your presence here today, but also for your service as Surgeon General of the United States. I can't think of another person who is in Federal service with whom I have looked to with more pride you and the job you have done for the United States of America.

Dr. NOVELLO. Thank you.

The CHAIR. Thank you very, very much.

Mr. TRAFICANT. Thank you, Mr. Chairman. I now recognize the former mayor from Compton, California, Mr. Tucker.

Mr. TUCKER. Thank you very much, Mr. Chairman. First of all, I want to say that I am very happy to be here this morning. There is a lot of activity going on on the Hill at another subcommittee within Small Business, but I am happy to have some time here.

I want to applaud you, Mr. Chairman, for the leadership that you have taken in this subcommittee. Obviously you told us we were going to be real busy in this subcommittee and you are a man of your word. Already we have had two bills to go on the House Floor. I think this is probably the most active subcommittee in the whole House thus far.

But Dr. Novello, we appreciate your being here. Obviously this hearing process is going to be very important to Americans all over America who are concerned about health care and health risks. I encourage the committee to be open-minded as we balance, as you have indicated, the interests of smokers versus nonsmokers.

I would imagine it would be fair to say—it seems as though you have already said it—that your concern is not so much to be on a crusade to penalize smokers or to shake their behavior, but to support the rights of nonsmokers and that if in fact as an indirect consequence of that, people reduce their smoking, then so be it. Would that be a fair statement?

Dr. NOVELLO. Yes.

Mr. TUCKER. All right. One of my questions to you relates to a point that you made earlier, Madam Surgeon General. You mentioned—I think you were talking about the association of any possible diminution as it relates to the labeling on the packaging. And I think you made some very cogent points as to that, just kind of a separate issue, but nonetheless related. You made a comment about African-American youth decreasing in their smoking practices or habits.

Is that directly linked to the labeling process or do you have any information as to why that is occurring or is there data on that?

Dr. NOVELLO. I can tell you that perhaps Dr. Erikson might be able to give us details. But I was so proud and so pleased to see that, even though the adolescents kind of flip here and there have not gone as high as I wish, the African-American youth has dropped from 21 percent to 5 percent. That is quite a statistic and I wished that was well known all over this country.

But we also find that with African-American adolescents brands that they prefer are the mentholated ones and when that is the case, we have to look into what advertisements for our neighborhoods, specifically in minority neighborhoods where 90 percent of all the billboards are touting behaviors that, when you do them, it is distinguishing to be this.

One has to look into this. And those statistics we don't hear too much, but it makes quite a difference in the life of people who want to do well for themselves.

Dr. Erikson, do you have any other data?

Mr. ERIKSON. Just to reinforce, it is a success story among Los Angeles high schools where they dropped from about 20 percent smoking which was white teenagers to 5 percent among Blacks, as the Surgeon General said. We don't understand exactly why that happened, but we know it is real and it is a success story and we need to know more about it so we can protect it and see if we can transfer it to the white teenagers.

Mr. TUCKER. Thank you very much, both you doctors.

Thank you very much, Mr. Chairman.

Mr. TRAFICANT. Thank you, Mayor. The Chair now recognizes the gentlewoman from Texas, Ms. Eddie Bernice Johnson.

Ms. JOHNSON. Thank you very much, Mr. Chairman. I will be brief in my remarks.

Being from the health caring professions, it is difficult to rationalize and make excuses for the effects of smoking on our health care system and the cost of it, and in the lives of people.

While none of us are interested in putting industries out of business that we have relied on for jobs, this is a difficult time in our country where we must reduce the cost of health care. The only way we can do that is to make some bold and tough decisions. This is one of them because the more smoking the more costly it becomes for the persons who pay the health care bill. We will just be paying more.

We recognize smoking as an acceptable addiction in our society. It is probably the toughest addiction that we have experienced because it is a legal addiction. But we have got to bite the bullet. We have got to save lives. And we know that it is detrimental to life and we have got to cut the health care costs because we know the costs of lung cancer and circulatory diseases.

I have suggested and I shall continue to suggest to the industry to rev up that R&D and try to find ways to save industry by producing, in some fashion, medications for treatment. Perhaps we should try to lean less and less on the sale of cigarettes which are not altogether tobacco.

I think we will reduce our health care costs a tremendous amount and not to say how much it will reduce suffering from lung cancer and heart disease and circulatory problems. We simply must bite this bullet. It is an essential time in our history. Change is hard, but change has got to come. People are demanding it now. And we have got to make some tough choices, not against any industry, but on behalf of the American people.

Thank you.

Dr. NOVELLO. Ms. Johnson, I could not have said it any better.

Ms. JOHNSON. Thank you.

Mr. TRAFICANT. I thank the gentlewoman from Texas in summarizing her questions briefly here.

Doctor, you do support a total ban in the Federal workplace, that which we have jurisdiction over; is that correct?

Dr. NOVELLO. I would say as a scientist and as a pediatrician, as a doctor who sees people every single day, I would say it would be something that should be looked into very closely. Remembering that we are not anti-smokers, that we are anti-smoking, so that we are respecting the rights of smokers, we do not forget to respect the rights of nonsmokers as well.

Mr. TRAFICANT. We thank you very much. You have a tough job. We subsidize tobacco growers, but we tell people it kills them. Congress works in unusual ways. Maybe you could help us develop a leadership role because I think we usually follow or just simply get out of the way. We need your help. I thank you very much, to you and Dr. Erikson. We thank you for being here.

Dr. NOVELLO. Thank you.

Mr. TRAFICANT. With that, I would like to welcome a panel of two very distinguished Members of Congress, both leaders with legisla-

tive experience in many different issues. The first is the Honorable Richard J. Durbin of Illinois, a leader in the fight to take on tobacco and to help reduce problems associated therein. And the Honorable Romano Mazzoli of Kentucky.

But before I would introduce you for your statements and ask you to summarize, I would like to recognize the Chairman of the Public Works Committee, for his statement or any remarks.

The CHAIR. Mr. Chairman, am I limited in terms of the time you have granted me?

Let me first of all thank you, Mr. Chairman, for your leadership on this issue. I really want to commend you in terms of your own foresight and your very strong leadership in convening this hearing and bringing to the surface this subject matter.

Of course, I also want to commend our colleagues Mr. Durbin and Mr. Mazzoli in terms of what they have done so far in the 103rd, but more importantly, if I might say thanks to Mr. Durbin for what he did as it related to smoking on airplanes. It was during the time that I chaired the Aviation Subcommittee that Mr. Durbin was able to do that. I just want to commend you for your very, very strong leadership.

Mr. Chairman, there is no question that the issue of smoking in Federal buildings is one that needs our very thorough review and action. In fact, the report issued by the Environmental Protection Agency at the beginning of this year makes this hearing all the more timely. Now, it is not too often that I share the fact that I am a former smoker and a lung cancer survivor. I had a lung cancer operation in 1983, and as a result, my health is now very, very good.

But quite frankly, given my own personal experience, I am especially alarmed by the EPA report that categorizes the effects of secondary smoke as a Group A carcinogen. Group A carcinogens include substances such as benzene, radon, and arsenic.

Now, this new information raises two important issues. First and most obvious is the threat to the health of those who work in the buildings. We have a responsibility to provide them with a safe working environment. Does this latest report mean that to provide that safe working environment we must provide a smoke-free environment?

Second is the question of legal liability. Now that we have this report from EPA, if we do not ban smoking in Federal buildings, do we become liable for any health problems that are suffered by nonsmoking employees working in Federal buildings where smoking is allowed?

In a legal sense, once we know that there is a hazard and then fail to correct it, have we exposed the U.S. Treasury to damages for disability and for death? In short, the question here is where both our obligation is to protect our employees and our obligation to protect the taxpayers requires us to ban smoking inside Federal buildings.

Mr. Chairman, obviously these hearings are timely. These hearings are very important. So, again, I want to commend you and Mr. Duncan for holding these hearings, and I look forward to hearing what the witnesses have to say so that we may become more edu-

cated about the issue and come to some conclusion, hopefully, to determine what course of action we take in the future.

Again, Mr. Chairman, thank you very, very much.

Mr. TRAFICANT. I thank the chairman, and I would like to echo and reiterate many of those remarks. And I agree with him. I would like to thank Mr. Durbin for his leadership in that he had in fact advanced legislation regarding smoking on airplanes. I think it was a tremendous help to the American people. I commend him on that.

I understand there is an agreement on order and we will not go by seniority. We will recognize the gentleman from Illinois, the distinguished gentleman.

TESTIMONY OF HON. RICHARD J. DURBIN, A REPRESENTATIVE IN CONGRESS FROM ILLINOIS; AND HON. ROMANO L. MAZZOLI, A REPRESENTATIVE IN CONGRESS FROM KENTUCKY

Mr. DURBIN. Thank you, Mr. Chairman. First, let me thank you for this hearing. I am really grateful to have you as an ally. Despite your reputation of being shy, reserved, and soft spoken, I sense you have a certain passion for this issue which will help bring this message to the American people and I commend you for this hearing.

I also want to thank my colleague, Congressman Mazzoli. He has shown extraordinary political courage in making public his views on the importance of sensible regulation of smoking. It is a more difficult political decision for him than many, and I am happy to say that he has been courageous and steadfast in standing up for this important public health issue.

As has been said before, and I am sure Congressman Mazzoli will say again, we are not anti-smoker, we are anti-smoking, particularly when it harms innocent people.

Within the last few years, we have formed a Task Force on Tobacco and Health on Capitol Hill. I am happy to have the chairman of this subcommittee as one of our newest members. We are more than 40 strong, Democrats and Republicans, who are publicly advancing this important public health agenda, and I hope some members of this subcommittee, particularly Congresswoman Johnson who has a background in public health, will consider joining us in making public our concern on this issue.

We have some questions to ask this morning, but the most fundamental question to be asked is this: Why is the Federal Government lagging behind State and local governments in moving towards sensible smoking regulation? Are Federal buildings somehow safer? Are Federal buildings inherently designed to keep secondhand smoke from those who are innocent?

Are Federal employees immune to the dangers of secondhand smoke? Do Federal employees care less about the dangers of this particular hazard than other government employees? Are Federal agencies—I will follow up on what the chairman has said—are Federal agencies more likely to be exempt from workers' compensation claims relating to lung damage from secondhand smoke?

The answer to all of these questions is obviously no. There is only one reason why the Federal Government is lagging behind

State and local governments in regulating smoking in public buildings. The clout of the tobacco lobby.

Today, even in this hearing room in the seats, and lining the wall also behind me, are the lobbyists for the largest tobacco companies in America. It is their constitutional right to be here. I would fight for that right for them to express their point of view, but let us not diminish at all the impact they have on public policy in the Federal Government.

Federal leaders have been cowed by the mighty tobacco giants with their PAC funds, with their intimate involvement in financing political caucuses and activities on Capitol Hill, both Democratic and Republican, with their phony smokers' rights groups, with the threats they make to their political opponents.

And you need only reference our friend in Oklahoma, Congressman Synar, who they targeted for defeat in this last election; and fortunately they failed. You can understand the clout of this lobby and why the Federal Government lags behind most other governmental units in the United States. We shrink away from our responsibility.

In response to these situations, I have introduced legislation, H.R. 710, to protect Federal employees from secondhand smoke in the Federal workplace and particularly to protect children from secondhand smoke while they participate in federally funded children's programs.

How can we, in good conscience, subject a child coming in for the WIC program even to the possibility that an employee will be smoking in their presence now that we have the clear evidence from the EPA report about the dangers of secondhand smoke?

I am pleased to have more than 40 cosponsors for this measure, including Congressman Mazzoli and Congressman Hansen who has also been a leader.

Mr. Chairman, with regard to Federal workers, the bills you and I have both introduced have the same goal, the protection of Federal employees who count on us to provide a safe environment as they serve the American people.

Let me say that I think it is important for us to put a perspective on this that was mentioned earlier. We genuinely have to help those smokers who want to quit, particularly Federal employees. One of the insidious propaganda campaigns from the tobacco lobby is that suggestion that anyone can quit smoking whenever they want to. We all know better from our personal experience. It is a terrible addiction.

I have seen people who have gone through open heart surgery because of their smoking habit or who have faced cancer surgery, and in the hospital—I have seen this personally—in the hospital, when they can finally hobble away from the intensive care unit, start smoking again. It is a terrible addiction.

We cannot just say, "Go, smoke-free and be on your own." We have got to stand behind the Federal employees who want to quit. We have got to help them and I think that is an important element as we consider this issue.

The next point I would like to make is this. We have come a long way. In July of 1987 when I brought to the Floor of the House of Representatives the legislation to limit smoking on airplanes, we

won by five votes and nobody gave us a chance. It was the first time we confronted this giant tobacco lobby in terms of regulating smoking. And we won.

For those who weren't present, you should have heard the screams from the other side. There was going to be fighting in the aisles on the airplanes over America. All of the flight attendants had to be prepared for these violent confrontations with smokers who would never adjust to the idea that they couldn't smoke on airplanes. Hogwash.

The facts have come out through the FAA: we have fewer than one complaint per million passengers in America about smoking regulation on airplanes. And I think that should be a model for us when we try to establish a standard for the Federal workplace—that, in fact, most people, even smokers, will go along with sensible smoking regulation.

But let me say, Mr. Chairman, we have a long, long way to go at the Federal level. And if you wonder whether we do, I ask each of my colleagues on this subcommittee to just look in front of you right now. Look at what's sitting in front of you on your desk right now. An ashtray. This is an anachronism, that in a room like this, with so many nonsmokers, we have an invitation for Members of Congress to smoke.

I suggest to you that in a short period of time, the presence of ashtrays in this kind of setting will be as likely as the presence of a spittoon was when we first came to Congress. These days are over, ladies and gentlemen, these days of inviting people to smoke without any concern for the audience and other Members around them. I would hope that this subcommittee and full committee would take the initiative of making their hearing room smoke-free. We are going to be doing it in the subcommittee room that I am presiding over upstairs.

We have a long way to go. It has to include all of the Federal Government. We cannot exempt Capitol Hill. We should go smoke-free and set the standard for the rest of the Federal Government. I commend the chairman for his leadership on this issue and thank you for the opportunity to testify today.

Mr. TRAFICANT. I just want to say, hogwash! Strong language today from this great leader in our Congress. The issue has changed significantly now with this Class A group.

We are not going to have questions at this time. We are going to move to Mr. Mazzoli, but I would like to say this. You are not implying that there be an amendment to this bill to ban ashtrays in the Capitol?

Mr. DURBIN. I hope to make them unnecessary.

Mr. TRAFICANT. Tremendous. We really appreciate your leadership and your testimony here today, Mr. Chairman, and we appreciate your being here.

The next gentleman, and I think Chairman Durbin had made a statement that he comes here at some political risk but that is the kind of guy that he is, and there are members on this subcommittee that face political risk. I want to commend the Ranking Member of this committee, Mr. Duncan from Tennessee, for his strong leadership and willingness to look at this issue that faces the

American people and also the subcommittee members especially, also Mr. Clyburn and all of us.

I appreciate that and I would say that Mr. Mazzoli has been a leader. He tries to do what is right and we are proud to have you here. Mr. Mazzoli.

Mr. MAZZOLI. Thank you, Mr. Chairman. I appreciate that. I thank your subcommittee for giving us this time. Let me salute my colleague and dear friend, Dick Durbin, for his extraordinary leadership. I remember, Dick, vividly that five-vote margin and I remember the very Armageddon-like statements that were profoundly said on the House Floor, and I think as we go back to those days when simply, if I recall correctly, there was a limit of flights of two hours or less and now we have continental bans. And there are many airlines that are thinking of global bans. We think how could anyone have not been for that kind of bill.

So I think, Mr. Chairman, because of your leadership and the high quality of your subcommittee and the statements made by our dear friends from San Jose, the gentleman who chairs the full committee, I think maybe five years from now we will look back on this hearing and say how could we have ever had a serious problem of banning the use of tobacco in Federal buildings. It will be that simple.

Let me make a few points and then any questions from our colleagues. Let me salute the high quality of the questions that happened when Mr. Durbin and I were in the room through most of the questions. I really was struck by the insightfulness of your panel. I am sure the work product of this committee will reflect that kind of insightfulness and leadership on your part.

It is true and I think maybe Mr. Duncan was one who first brought up the issue, this is not to be against the smoker. This is to be against smoking as a health hazard, as a matter of inconvenience and discomfort to a lot of people. And because it is that, we do have a responsibility for smoke cessation programs. We have a responsibility for conversion programs, as the one we have for defense workers, for tobacco workers like in Louisville and in Kentucky and tobacco farmers we have in Louisville and other States of the Union. So as we make these moves we do have additional responsibilities.

I was struck again by what Mrs. Johnson said, our new member from Texas, about the health care cost. The easiest way we can cost contain in the health care field is to try to prevent people from getting sick in the first place. Ergo, having fewer people smoke means you have fewer people getting sick and less cost.

So there is a clear relationship. The former Mayor of Compton, our new Member, Mr. Tucker, brought out from the Surgeon General an interesting line of questions and that is look at our inner cities and where the ads are, where the bill boards are and what they display. Among other things that infamous Joe Camel. I have taken my little campaign to the Floor of the House of Representatives about Joe Camel and that clear and unequivocal direction of that campaign towards young people, not necessarily people even who are smokers. I don't think it is brand switching as much as causing people to smoke in the first place. As well, there are other substances deleterious to health in this situation.

So this panel is on to a very interesting multifaceted subject. I think the questions Mr. Durbin raises are difficult but soluble by your committee. I am proud to associate with Dick Durbin, proud to associate with the Surgeon General who has been a woman of great courage to take on this whole issue, and I am glad that the gentleman from California, Mr. Mineta, our full committee Chairman, did make mention of that while he was in the room.

I am proud to be a co-sponsor of the pro-kids bill which Dick has developed, which does deal with youngsters in Federal settings. I might say that it is awkward for me to be here at this time and not because of what my dear friend has said about me and I appreciate those comments and I hope they are deserved. But because when we in this committee act, we are causing our friends, our colleagues in some cases and certainly Federal workers inconvenience and discomfort. It may mean that they will have to leave their workplaces temporarily if they wish to smoke.

I don't do that casually. I do not do that as a hair shirt. I don't do that as a reproach or example. I join others in doing that in order to sublimate what I consider to be rights to other greater rights. There are rights of smokers; there are rights people have to stay in the workplace and not have to go out into the cold. But there are greater rights, I think, and the balancing of these rights is a constant responsibility of Members of Congress. We do it all the time.

I think that here we are sublimating these rights because there is a national trend in that direction. In a way, the Federal Government can still be a leader, but we are going to be, as Dick suggested, a kind of follower because other States, like California, where the gentleman from Compton is from, has through executive orders banned smoking in most of the Federal buildings and State-owned buildings in California. The Orioles up the road in Baltimore will have a no-smoking policy in their outdoor stadium. Notre Dame, and I hesitate to bring that up with my friend from the University of Pittsburgh, but I will have to say at Notre Dame Stadium they don't have smoking in an outdoor bowl. Maybe the next time the Panthers come in maybe we can overcome you in different ways than with smoke.

Governor Jones in Kentucky, Mayor Jerry Abramson and County Judge David Armstrong in Jefferson County are trying to do the right thing in setting up smoke-free areas. Why this trend? I think Mr. Mineta brought it up. Not only is there mounting evidence of a health connection to smoke, EPA reported January 7, linking it to ETS or downwind smoke, but we have the profound possibility of multibillions of dollars of legal liability to the Federal Treasury, to the treasuries of the 50 States and the five territories and to corporations and companies galore if something isn't done relating to this EPA report.

I would mention one statement I heard recently. On the 8th of January, the day after the EPA study came out, I was in Louisville conducting a meeting with the Chamber of Commerce and when the Q and A part came a young owner of a company stood up and said, "Mr. Mazzoli, this EPA report of yesterday really has me scared because I am worried about my liability in the workplace. I am really afraid."

I said, I honestly believe you have a lot to be afraid about because I think, in the last analysis, the dollars and cents argument, may well overwhelm all the other arguments, including the social consciousness, and the fact that our children are encouraging us in each case not to smoke.

We can challenge the reports methodology, the analysis and other so-called defects, but to me it is simply one of the last and one of the best and one of the most weighty and persuasive arguments that could be made in favor of the simple fact that smoking tobacco, if not harmful, which I suggest it is, is certainly not helpful to your health.

I think that we as people in Federal Government have a responsibility to our workers to protect them from something which is certainly not good for them if it isn't in some way absolutely bad.

I think it is time for the Federal Government to join the trend, to move to the lead, to set the example, to be the standard-bearer and I think in that setting, Mr. Chairman, the Capitol complex, and this is difficult, because we are talking about our colleagues, our friends, people that we work with day to day, people that we came with to Congress, ought to be smoke free. We have to be honest and objective and not hypocritical here, and we have to apply this to the Capitol complex.

My last statement is and I started with it, I salute you for the high quality of your committee work and in the process don't forget the Federal employees and tobacco workers and tobacco farmers.

Mr. TRAFICANT. I appreciate your testimony. I know Chairman Durbin had a couple other concerns and he didn't want to interrupt, but if you have to leave, Mr. Chairman, whatever your schedule is, if you want to stay you are more than welcome.

Mr. DURBIN. I will stay a few minutes.

Mr. TRAFICANT. I just wanted to say that things have changed. This issue is much different. I think both of you have been leaders in this issue.

We certainly may not be able to do some of the things that you are concerned with. We simply have jurisdiction over the public buildings and the workplace and rental space of our employees. But I would say that I don't know what legislative vehicle will ultimately be that will deal with this issue. We would be open to accommodating an open amendment process, those concerns of your leadership if, in fact, a straight ban just dealing with the Federal buildings is the issue the Congress decides to go with first.

I am not speaking for anybody else. I am just saying that for myself.

Second of all, it concerns me very much concerning the liability issue. I think both of you spoke to that. Are we going to see workers compensation cases coming in the future? We have very creative attorneys out there. There will be creative litigation. We can all sense that. What will be the position of our Federal Government after having, first, the report of the Surgeon General, then corroborating the report of the EPA notwithstanding the controversy that questions the data, but finally our government is telling our government we are in trouble.

We have a class A carcinogen on our hands and even Ronald McDonald started to take some issue with it.

Even though you are from Notre Dame, Mr. Mazzoli, I am not going to let you sneak that in especially with Durbin here.

Mr. DURBIN. Let me, Mr. Chairman, tell you one story that exemplifies our mutual concern. I received a letter recently from a gentleman I didn't know in the State of Mississippi far from my district in Illinois. He has a daughter who works here on Capitol Hill. She works on the Minority side for one of the committees. She shares an office with another woman and this woman insists on smoking all day long. This young lady, this daughter of a man who wrote me, has a blood pressure problem. He wrote in desparation saying we can't get this woman to stop smoking in the presence of my daughter. She says that this will aggravate her physical condition.

It doesn't take Clarence Darrow to figure out that if this young lady's physical condition is worsened and she can attribute any part of it to her exposure to secondhand smoke, the ultimate liability will not be against the personal assets of this other employee, it is going to be against the Federal Government and the Federal taxpayers who are going to end up paying some workers' comp claim.

This doesn't take a great leap in logic, to understand where we are today. And we have been forewarned by EPA. We are forewarned by people who were exposed to secondhand smoke of the personal dangers they face. It is time to wake up. This is a matter of dollars and cents and good health judgment, and I think, frankly, it is time to go smoke free to protect a lot of innocent people.

Mr. TRAFICANT. I think we have also been forewarned by two leaders of our Congress, Mr. Durbin of Illinois and Mr. Mazzoli of Kentucky, and that makes it more compelling, because even the Congress is telling the Congress you are exposing yourself, not like the old days, I would say, and with that I would like to move on down here.

I recognize the Chairman, Mr. Mineta, who finally came back because he wants to take you guys on.

The CHAIR. Mr. Chairman, let me thank you for your leadership and our two Members have spoken very well and very fully and in-depth. I have no questions.

Mr. MAZZOLI. Thank you.

Mr. TRAFICANT. That was easy.

Mr. Clyburn.

Mr. CLYBURN. No questions.

Mr. TRAFICANT. We have a vote on, but we can expedite and get over to vote and come back.

Mr. CLYBURN. I have no questions, Mr. Chairman.

Mr. TRAFICANT. Mr. Tucker.

Mr. TUCKER. No questions, Mr. Chairman, just to add my congratulations to these distinguished gentlemen for very courageous testimony before this subcommittee today. I think they embody the essence of leadership here on Capitol Hill. Thank you, gentlemen.

Mr. TRAFICANT. I just want to conclude with this group by saying one thing. There are not many lobbys in America that support me and you have probably taken the last one that was on the fence and thrown that out. Maybe that was your goal today, but we welcome your testimony. We appreciate your leadership. This after-

noon we are going to be asking you to help us in a consensus, some vehicle to accomplish our goals.

Mr. MAZZOLI. Thank you, Mr. Chairman.

Mr. TRAFICANT. Thank you for being here. We appreciate your testimony.

With that, I would like to say that maybe we will suspend, recess temporarily and we will reconvene at 10 minutes to the hour.

The meeting is recessed temporarily.

[Recess.]

Mr. TRAFICANT. The subcommittee will reconvene. I call to the table Dr. William H. Farland, Director, Office of Health and Environmental Assessment, U.S. Environmental Protection Agency. Doctor, we welcome you here and the names of your assistants, if you will, and if they will be participating. That is fine.

TESTIMONY OF DR. WILLIAM H. FARLAND, DIRECTOR, OFFICE OF HEALTH AND ENVIRONMENTAL ASSESSMENT, U.S. ENVIRONMENTAL PROTECTION AGENCY, ACCOMPANIED BY DR. HUGH McKINNON, HUMAN HEALTH ASSESSMENT GROUP, AND DR. STEVEN BAYARD, BIOSTATISTICIAN

Dr. FARLAND. I would like to introduce those with me. On my left is Dr. Hugh McKinnon. He is a physician and head of our Human Health Assessment Group. On my right, Dr. Steven Bayard, Steven is a biostatistician. He was the project manager for the report we will be talking about this morning. He is one of the principal authors of that report as well.

Mr. Chairman, and members of the subcommittee thank you for the opportunity to appear before you today to discuss H.R. 881, a bill to prohibit smoking in Federal buildings.

The U.S. Environmental Protection Agency (EPA) has recently published a major assessment of the respiratory health risks of passive smoking (Respiratory Health Effects of Passive Smoking). The report concludes that exposure to environmental tobacco smoke (ETS), commonly known as secondhand smoke, is responsible for approximately 3,000 lung cancer deaths each year in nonsmoking adults in the U.S. and seriously affects the respiratory health of hundreds of thousands of children. The following testimony summarizes the development of the report, the scientific review process, and the major findings. It concludes with EPA's endorsement of the objectives of the proposed legislation.

In recent years, comparative risk studies performed by EPA and its Science Advisory Board have consistently ranked indoor air pollution among the top five environmental risks to public health. Environmental tobacco smoke is one of the major indoor air pollutants and, given the known health impact of tobacco smoking, there has been concern that nonsmokers may also be at risk of serious health effects.

As part of its efforts to address all types of indoor air pollution, in 1988 EPA's Indoor Air Division requested the EPA Office of Research and Development to undertake an assessment of the respiratory health effects of passive smoking. It was prepared by my office, the R&D office.

The document has been prepared under the authority of Title IV of Superfund (The Radon Gas and Indoor Air Quality Research Act

of 1986), which directs EPA to conduct research and disseminate information on all aspects of indoor air quality.

A draft of this assessment was released for public review and comment in June 1990. In December 1990, EPA's Science Advisory Board (SAB), a committee of independent outside scientists, conducted a review of the draft report and submitted its comments to the EPA Administrator. That was in April 1991. In its comments, the SAB's Indoor Air Quality/Total Human Exposure Committee concurred with the primary findings of the report, but made a number of recommendations for strengthening it.

Incorporating recommendations from both the public and the SAB, a revised draft was transmitted to the SAB in May 1992 for a second review. Following a July 1992 meeting the SAB panel endorsed the report and its conclusions, including a unanimous endorsement of the classification of environmental tobacco smoke as a Group A (known human) carcinogen.

The EPA also received and reviewed public comments on the second draft and integrated all appropriate material into the final risk assessment. The final report was released in January 1993, at a joint press conference held by former Administrator Reilly and former Department of Health and Human Services Secretary Sullivan.

Based on the weight of the available scientific evidence, EPA has concluded that the widespread exposure to environmental tobacco smoke in the U.S. presents a serious and substantial public health risk.

In adults, ETS is a human lung carcinogen responsible for approximately 3,000 lung cancer deaths annually in the U.S. nonsmokers. ETS has been classified as a known human, Group A carcinogen under EPA's carcinogen and assessment guidelines. This classification is reserved for those compounds or mixtures which have strongest data to determine a cause and effect relationship including data from human populations.

Only 10 other agents, including asbestos and radon, have been classified by EPA as class A and ETS is the only one for which cancer has been observed at typical non-occupational environmental levels.

In adults ETS has subtle but significant effects on the respiratory health of nonsmokers including coughing, phlegm production, chest discomfort and reduced lung function.

In children ETS exposure increases the risk of lower respiratory tract infections such as bronchitis and pneumonia. EPA estimates that between 150,000 and 300,000 of these cases annually in infants and young children up to 18 months of age are attributable to exposure to ETS. Of these, some 7,500 to 15,000 will result in hospitalization.

ETS exposure increases the prevalence of fluid in the middle ear, a sign of chronic middle ear disease. Fluid in the middle ear is the major cause of hospitalization of young children for an operation in the U.S.

ETS exposure in children irritates the upper respiratory tract and is associated with a small but significant reduction in lung function.

ETS exposure increases the frequency of episodes and severity of symptoms in asthmatic children. The report estimates that 200,000 to 1,000,000 asthmatic children have their condition worsened by exposure to environmental tobacco smoke.

ETS exposure is a risk factor for new cases of asthma in children who have not previously displayed symptoms.

EPA reached its conclusions concerning the potential for ETS to act as a human carcinogen based on an analysis of all the available data including more than 30 epidemiologic or human studies looking specifically at passive smoking and lung cancer in nonsmokers.

In addition, EPA considered information on active smoking, animal data, biological measurements of human uptake of environmental tobacco smoke components and other available data. The conclusions on lung cancer were based on what is known as the total weight-of-the-evidence approach rather than any one study.

Specifically, the finding that ETS should be classified as a Group A carcinogen is based on conclusive evidence of the dose-related carcinogenicity of mainstream smoke and the sidestream smoke given off by the burning end of the cigarette and the known exposure and uptake of ETS at levels which could increase risk. The finding is bolstered by the statistically significant exposure-related increase in lung cancer in nonsmoking spouses of smokers which is observed in an analysis of more than 30 epidemiology studies, from eight different countries, that examined the association between secondhand smoke and lung cancer.

The weight-of-evidence analysis for noncancer respiratory effects in children is based primarily on a review of more than 100 studies available to us now including over 50 epidemiological studies of children whose parents smoke.

Mr. TRAFICANT. Doctor, since we have gotten through the bulk of the statement, could you summarize the remainder so we could move into questions for you guys, if that is possible.

Dr. FARLAND. I think it is important for us just to indicate the implications of this report to public policy. We put these perspectives around these risks associated with ETS. EPA estimates that about 20 to 30 percent of all lung cancers are associated—caused by factors other than a cancer patient's own smoking. These risks are high compared with other risks EPA deals with.

This report together with a report by our colleagues at NIOSH sets a firm scientific foundation for actions to reduce exposure of nonsmokers to tobacco smoke.

While we don't have any regulatory authority over ETS, we intend to work with other Federal agencies to ensure that this issue is adequately addressed.

In cooperation with other government agencies, then, EPA will carry out an education and outreach program over the next two years to inform the public and policy makers of what to do to reduce health risks of ETS as well as other indoor air pollutants.

I would like to personally recommend that the provisions of the bill we have before us today be extended to include a ban on smoking in all vehicles owned or leased by the Federal Government. Studies have shown that smoking in smaller confined spaces can dramatically increase ETS concentrations over those normally in homes or buildings.

To conclude, the general thrust of H.R. 881 represents responsible governmental action for protecting public health and will provide a model for State and local governments to follow.

EPA therefore supports the objectives of H.R. 881 while deferring to interested agencies regarding details in the bill.

We will be glad to answer any questions you may have, Mr. Chairman.

Mr. TRAFICANT. I thank you, Doctor, and would inform you that I do plan to offer an amendment to the bill that would ban smoking in those Federal vehicle fleets.

Rather than me go first, our distinguished ranking Republican member has another meeting and I would like to defer to him first.

Mr. Duncan.

Mr. DUNCAN. Thank you, Mr. Chairman.

I will try to be brief.

Dr. Farland, you may have been here a while ago and heard some of the questioning, but Mr. Emerson mentioned some scientist at Yale, I think, who said that your study was flawed but it was for a good cause so it didn't really matter.

I was not familiar with that quote or of that gentleman. Had you read that or heard that and what do you have to say in response to that?

Dr. FARLAND. There are several comments I might make, Dr. Feinstein has gone on record with respect to his feelings with that. As in all scientific issues there is a range of scientific opinion around this issue as well. We are familiar with his concerns and have met with him and have discussed those directly with him, as a matter of fact.

Our position at this point is that the general scientific community supports both the approach that we took to evaluating the data and the findings that we have made, and while there will be individuals who would not agree with the approach or the findings, we think we have a general consensus behind us on the positions taken by the agency in this matter.

Mr. DUNCAN. Who is Dr. Feinstein? I am not familiar with him. Is he a respected scientist?

Dr. FARLAND. I don't know his credentials well, Mr. Duncan. He is an epidemiologist from Yale, an academic outspoken on methodologies in epidemiology and has been widely publicized. He was a member of the panel that looked over early drafts of this document, the panel convened by R.J. Reynolds, I believe.

Dr. Bayard, any other comments?

Dr. BAYARD. Dr. Feinstein is, as I understand it, I knew him when I was at Yale. He is an M.D. at Yale. The quote that you ascribe to Dr. Feinstein was not his quote, but his quote quoting some other unnamed person that it was bad science but in a good cause, so let's let it go.

I guess Dr. Feinstein can speak for himself, but we have met with him. We met with him the other day and-last week, last Tuesday, and also in the meeting were members of the National Cancer Institute and these are real scientists and basically we disagree with him. He didn't bring up any issues that we felt were substantive at all.

Mr. DUNCAN. I am not sure that I am exactly clear on how you arrived at the precise figure of 3,000.

Why not 10,000? Why not 100,000? Why not 1,000? How did you do that exactly?

Dr. FARLAND. Mr. Duncan, risk assessment is an imprecise science. There is uncertainty in these numbers and we want you to understand there is uncertainty. There is uncertainty with respect to the statistical approach we used and uncertainty with respect to susceptibility of individuals in those sorts of things.

This is addressed specifically in the report. There is a range around that number based on statistics of 400 cases to 7,000 cases. There is a discussion about——

Mr. DUNCAN. Your actual study came up with a range of 400 to 7,000?

Dr. FARLAND. That's right. That was the statistical range, around that number. We did a separate analysis of one of the most powerful studies that recently became available to us, the Fontham study, and using statistical methods to do that particular study we came up with an independent estimate of approximately 3,600 cases and so in the report there is a discussion about the uncertainty associated with that number trying to put that in perspective, but a statement that we are reasonably confident of is that a number of 3,000 cancer deaths attributable to ETS in nonsmokers is a good estimate.

Mr. DUNCAN. Let me ask you this. As you may have heard me say, I don't smoke, I don't like smoking, I try to encourage people not to smoke and I don't want to imply or seem like I am trying to defend smoking, because I am not. I do want to make sure that we do look at this fully and what I am wondering about, in the past few years we have seen so many scientists who seem to love headlines that they have come out with all sorts of things and the general public is out there now saying, well, they tell us everything kills us at some point or another.

I remember, I think it was "Nightline" or one of the shows had something a few years ago about peanut butter caused cancer. We had the alar scare and that turned out to be false and they say the asbestos, that we would be better off-you know, we spent billions removing it and we would have been better to have left it alone.

What could I say to someone who says that to me? What would you say to someone who says, well, this is just another of those scare things designed to encourage people not to smoke. It is not really accurate or not really fair. The study was not a broad-based study. That type of thing. What would you say to that?

Dr. FARLAND. Mr. Duncan, our business in my office is to do these risk assessments on lots of different chemicals and agents. This particular one is unique. It is unique because of the size of the database that we have available to us, the number of studies on human populations that are available to us, the unique situation that these studies have found in effect at environmental levels. These are positive responses in spouses of smokers who are exposed in their homes, not under occupational situations or high exposure levels or basically not a situation like other chemicals that we deal with where we must rely on toxicological information from studies in animals.

Here we have human information, 30 studies looked at for the cancer issue, a large number of studies, over 50 in children, humans, for the respiratory effects. This is a very different case from your day-to-day chemical carcinogen of the week, or whatever term the press might want to use on those types of situations.

We think we have an excellent database to work with and we have done the best job that we can do to evaluate that database.

Mr. DUNCAN. Finally, let me ask you this. There was an internal EPA report that really was dealt with on another issue, but it is entitled, "Credible Science, Credible Decisions, Safeguarding the Future".

It was very critical of the science that the EPA has used. For instance, it says among one finding, EPA has not clearly conveyed to those outside or even inside the agency its commitment to make high quality science a priority."

There are many other criticisms, but I don't want to take all that time now.

Are you familiar with that and do you think that any of those criticisms of EPA—and there are quite a few in there—do you think that any of those could be fairly applied to this particular study?

Dr. FARLAND. Again, Mr. Duncan, I am familiar with the report and certainly endorse the Administrator's suggestion that an independent panel look carefully at the science that EPA does. It has some very big implications for our Nation in the way that we deal with environmental issues.

I agree with some of the findings in the report. There are both criticisms and praise provided in that report for the types of things that we do. I think that the document, the review process that we have gone through, the open nature of the discussion that has gone on with this report really supports the approach that Mr. Reilly has suggested.

The fact is that this particular report should be an open discussion in the scientific discussion community, that it should represent the best science available and should be critiqued by a broad range of knowledgable people who have no real issue to gain with respect to the Environmental Protection Agency.

Mr. DUNCAN. Is there any safe level? Do you really feel personally that—do you feel personally that cigarette smoking should be made illegal?

Dr. FARLAND. Again, an opinion as a scientist and public health professional, risk is a matter of both hazard and exposure so that there will always be diminishing risks with diminishing exposure. At some point the risks will be so low as to be not meaningful anymore.

On the other hand, in this particular case, we have data from human studies in homes with smokers that provide a positive response; that is, lung cancer in those spouses, that is measurable. Epidemiology is a crude tool and because it can find that response, the fact is that responses are likely to be out there among people who are exposed at environmental levels.

I would not argue that there is no level that is not safe because safe is a judgment, but I would argue that the levels ought to be reduced to the extent possible.

Mr. DUNCAN. Well, thank you very much.

Thank you.

Mr. TRAFICANT. Thank you, Dr. Farland, for the straightforward answers. I have a few questions.

Number one, I would like the Fontham study that you cited.

Dr. FARLAND. Fontham study, yes.

Mr. TRAFICANT. That would be submitted into the record, and the Blue Ribbon Panel Report that critiqued the EPA findings, that also would be submitted for the record.

Dr. FARLAND. Yes.

[The following were received from Dr. Farland, and may be found in the record following Dr. Farland's prepared statement:]

Article: "Lung Cancer in Nonsmoking Women: A Multicenter Case-Control Study".

EPA Science Advisory Board Review of the Environmental Tobacco Smoke Risk Assessment—Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders.

Report of the National Institute for Occupational Safety and Health (NIOSH) Bulletin: "Environmental Tobacco Smoke in the Workplace (Lung Cancer and Other Effects)".

EPA Fact Sheet: Respiratory Health Effects of Passive Smoking.

Mr. TRAFICANT. What is the policy on smoking at your building?

Dr. FARLAND. We have had a smoke-free building for two years now, Mr. Chairman.

Mr. TRAFICANT. Smoke free?

Dr. FARLAND. Yes, smoking is allowed outside the building.

Mr. TRAFICANT. Only?

Dr. FARLAND. Yes.

Mr. TRAFICANT. What about nationwide? What do you do in your regional offices?

Dr. FARLAND. I believe that that is a region-by-region decision. I don't know the answer. I could get you the answer to that question, but I personally do not know the answer to that.

Mr. TRAFICANT. What has brought about probably significant and swift response to this matter is that there have been corroborating types of studies that are showing not only risk but significant liability problems. Sometimes the government works in strange ways.

But when you have a certain general finding and then you have other reports in the private sector through other groups, now you have the Environmental Protection Agency, the arm of the Federal Government, comes out and says that ETS is a class A carcinogen. It is a different ball game. Class A carcinogen.

Would you briefly tell me why it was designated class A? What are the standards of classification when looking at some of these hazards and to a greater or lesser degree what other types of substances or problems exist that have been classified perhaps at higher or lower levels, if you can get into that area for me.

Dr. FARLAND. Sure. I will try to be brief and again if you need to have follow-up questions, I would certainly be able to go forward with that.

In 1986 the agency published its risk assessment guidance that actually laid out the scheme for classifying carcinogens. It broke carcinogens into category A, known human carcinogens; category B, probably carcinogenic to humans; and category C, possibly carcinogenic to humans, chemicals that were of an inadequate database to

make a decision; and then category E, chemicals where it was known that they were not carcinogenic to humans, that adequate testing had been done to reach that conclusion.

Mr. TRAFICANT. In that regard, ETS, secondhand smoke, was evidently considered to be and is, in fact, a known carcinogen by the status of the class A rating.

Dr. FARLAND. That's right. The basis for it being a known carcinogen is the epidemiology studies that had looked directly at passive smoke, ETS, by evaluating primarily the spouses of smokers, nonsmoking spouses of smokers who are getting exposure to ETS while in their homes.

In addition to that, it took into account the known lung cancer effects of active smoking. It took into account the fact that the constituents of both active and passive smoking are similar. There are some 43 known and probable carcinogens found in that smoke.

It took into account animal studies that demonstrated the ability of tobacco smoke and its constituents to impact biological processes that lead to cancer in certain laboratory studies and it took into account a full range of studies that looked at the issues of levels that individuals are exposed to through their day-to-day activities in the environment whether they are smokers or nonsmokers.

Mr. TRAFICANT. In that regard would you name other class A carcinogens.

Dr. FARLAND. We mentioned radon, benzine, arsenic. In our integrated risk information system, the consensus database that the EPA uses, we identify a total of 10. I would be glad to submit that list to the committee.

Mr. TRAFICANT. I would ask that you have that list submitted to us.

Asbestos is one of those.

Dr. FARLAND. Certainly.

Mr. TRAFICANT. To your knowledge what has been the liability factor and what has been the status of litigation surrounding asbestos and the government's response, just briefly.

Dr. FARLAND. Unfortunately, that is not my area as a scientist. However, I am aware of much of the issues that have been coming up with regard to asbestos litigation. I think there is a general sense that the findings from the scientific community that asbestos is a known human carcinogen has absolutely stood the test of time. We have adequate data to suggest that humans who are exposed are at risk.

The question becomes those low levels that we were talking about earlier.

Mr. TRAFICANT. In your building you have a smoke-free environment. Did you consider the prospects of or the creation of retrofitting it towards a ventilated type of sequestered smoking room or smoking section?

Did you consider the prospects of or the creation of the retrofitting towards a ventilated type of sequestered smoking room or smoking section?

Dr. FARLAND. The individuals who are involved in the Indoor Air Division of our air program have been looking into those issues specifically, and they are developing reports related to that.

In our own building, the decision was made to go forward with, first of all, in the earlier days, a designated smoking area. It was found that that was not adequate because the smoke was coming into the general circulation and was still getting into the general workplace in the building. And so the decision was made, at least in our headquarters building, to go forward with a policy of smoking only on the outside of the building.

Mr. TRAFICANT. Do you have any costs associated with that scenario?

Dr. FARLAND. I am afraid I am not familiar with that particular issue.

Mr. TRAFICANT. Let me ask you this. You just designated an area within the building, but it was the same system, air circulating system, that—and there was no special retrofitting of this space for special types of ventilation, is that correct?

Dr. FARLAND. That is correct, and that was deemed not to be satisfactory.

Mr. TRAFICANT. What is meta-analysis?

Dr. FARLAND. Meta-analysis is a technique that is applied often to epidemiology studies to attempt to take individual low powers to detect an effect, and, if conditions are correct for combining them, combine them to increase the power to detect effects and to give a more robust understanding of the responses that might be seen in those studies. It is a relatively new technique that has been applied to clinical trials, not epidemiology studies but clinical trials in the pharmaceutical industry and in the drug industry, permitting response activities of the drug.

And under those conditions where one has very similar types of studies, meta-analysis can very easily be used to combine results of independent studies. It is more difficult to do when one has investigations that have been done by lots of different investigators, by slightly different types of studies and slightly different protocols.

So what we have done in our case is to take the results of studies that are a substantially similar design, that looked at similar end points and to lay out the pros and cons of combining those studies, doing a number of different analyses, both independent studies of the individual data as well as the combined study as well as a number of statistical approaches to all of the studies available to us and use that as part of our discussions surrounding the cause and effect relationship between ETS and lung cancer in adults.

Mr. TRAFICANT. What does statistically significant mean and what records can that be compared to or evaluated according to your answer here on meta-analysis?

Dr. FARLAND. I am going to defer to my biostatistician in just a moment here, but statistically significant is an opportunity for us to define our certainty about a particular finding in some study or some result.

In the case of epidemiology studies, we want to determine whether or not we have 90 percent certainty that the finding, the relative risks or whatever the finding of the study happens to be is not due to chance.

Mr. TRAFICANT. I hate to interrupt you, but there are those naysayers that are now saying there is a 95 percent standard, and the 95 percent standard had been utilized in many of your other

determinations and that the ETS report and factual data, as released, was released on a lesser standard or burden, that being of 90 percent.

How do you respond to that specific challenge that has been made that EPA has more or less lowered the standard on this particular ETS issue?

Dr. FARLAND. Very directly, we have not lowered the standard in this particular issue. The approach that we have taken is consistent all the way through the development of this report. We have used a 90 percent confidence interval, which is consistent with a technique called a one-tailed test of significance that has been used throughout the report.

While some of the individual studies have used 90 percent and others have used 95 percent, we have normalized them to 90 percent. It becomes a matter of whether you want to be 90 percent confident or 95 percent confident that this particular finding is not due to chance in the face of all of the other evidence that we have on active smoking and the animal studies and all the other things I ticked off.

Let me ask Dr. Bayard.

Mr. TRAFICANT. If you could, briefly. I have a question for you, and I don't want to lose track of that.

Dr. BAYARD. Sure. The way we are loosening our estimates or loosening our standards, I think, is the boldest concept. Basically, we usually use upper-limited estimates in deriving the 3,000 estimate. We all three made conservative assumptions. So we feel that estimate might actually be a bit low.

With respect to 90 percent versus 95 percent certainty, the concept is, basically, there is so much evidence from active smoking with dose response at very low levels with no evidence of a threshold that when you go to make your test on passive smoking, which is done at environmental levels, it is going to be a smaller dose. And you want to have a certain degree of certainty of picking up an effect if it exists.

So do you want to be 95 percent certain or 90 percent certain, given everything else you know about the dose response relationship of active smoking down to very low doses with no threshold?

Mr. TRAFICANT. So, Dr. Bayard, you are the scientist, I assume, that participated in the EPA findings and report; is that correct?

Dr. BAYARD. That is correct.

Mr. TRAFICANT. Are you telling the Congress that this passive smoke not only endangers the health of those workers in the Federal workplace but could lead to their death? Is that what you are telling us today?

Dr. BAYARD. Yes.

Mr. TRAFICANT. And if you were called in before a court of law on a contentious piece of litigation concerning this matter and the defense attorney said to you, did you, in fact, take every measure possible to notify the Congress of this danger that has produced this problem with my client, how would you answer that?

Dr. BAYARD. If I haven't, I will call everyone up tomorrow. I am trying now, and I would be glad to talk to anyone who wants to.

Mr. TRAFICANT. Do you believe that EPA now has not only sent out a guesstimate but is telling the Congress, you have a problem

on your hands. We don't have a question any more. We are not involved in the controversy here. It is our findings that we are to you, Congress, that this is a significant problem that you have in the workplace and to do something about it.

Dr. BAYARD. We believe the evidence is conclusive.

Mr. TRAFICANT. Dr. McKinnon, same question.

Dr. MCKINNON. Yes, sir. As a physician trained in public health and environmental medicine, I would say it is absolutely conclusive.

Mr. TRAFICANT. Dr. McKinnon, in closing here, there have been some discussions about $10 billion, $15 billion, $30 billion, $65 billion, guesstimates as high as $100 billion. Here is a policy that subsidizes tobacco and warns people that tobacco smoke kills them and now says the secondhand passive smoke could kill a nonsmoking neighbor or relative. Do you think any of those policies makes sense in light of the documentation that EPA has brought forward?

Dr. MCKINNON. Mr. Chairman, I think those policies are clearly in conflict. I understand, from my knowledge of American history, where those policies have come from and the role that tobacco growth has played in the formation of our Nation, but I think the time has come to do away with any encouragement for people to smoke or otherwise use tobacco products.

Mr. TRAFICANT. Would you support a ban on smoking in the Federal fleet?

Dr. MCKINNON. Yes, sir, I would.

Mr. TRAFICANT. Dr. Farland.

Dr. FARLAND. Yes. Absolutely.

Mr. TRAFICANT. Dr. Bayard?

Dr. BAYARD. Only in closed spaces.

Mr. TRAFICANT. In the fleet?

Dr. BAYARD. You mean vehicles?

Mr. TRAFICANT. Yes.

Dr. BAYARD. Yes.

Mr. TRAFICANT. Mr. Clyburn, do you have any questions for our panel from the Environmental Protection Agency?

Mr. CLYBURN. Well, since I did not hear the direct testimony, Mr. Chairman, I will waive any questions until the next hearing.

Mr. TRAFICANT. I would just like to say this, that you are charged with an awesome responsibility. Your report is coming under fire. There will be tests, studies, differing types of professional investigations and an awful lot of scrutiny into your findings.

One thing that Congress wants to determine is that the reliability of the evidence that is coming forward on the secondhand smoke is without question, certain, and the probabilities of correctness absolutely very high. And you are telling us that you are standing by your report.

Dr. FARLAND. Mr. Traficant, you have our testimony today. Our report is a matter of public record. We have had a large number of scientists review this report and the Scientific Advisory Board has unanimously agreed with our conclusions. We feel very strongly about the findings of this report and feel it will stand the test of time.

Mr. TRAFICANT. Well, I would welcome any and all documentation you have from any other professional scientific groups that have participated in or have performed collateral type of studies. I ask unanimous consent that such documents be sent to us.

I want to thank you very much. I appreciate your testimony, and I wish you the very best.

Dr. FARLAND. Thank you.

Dr. MCKINNON. Thank you.

[Subsequent to the hearing, additional questions on behalf of Representative Emerson were submitted to EPA. The questions and responses follow:]

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

July 1, '93

June 28, 1993

OFFICE OF CONGRESSIONAL
AND LEGISLATIVE AFFAIRS

Honorable Norman Y. Mineta
Chairman
Committee on Public Works and Transportation
U.S. House of Representatives
Washington, D.C. 20515

Dear Senator Mineta:

Attached, for insertion in the hearing record, are the United States Environmental Protection Agency's responses to questions which you forwarded to Dr. William Farland following the March 11 hearing on H.R. 881. These responses were prepared by EPA's Office of Air and Radiation and Office of Research and Development. Please feel free to contact me if we can be of any further assistance.

Sincerely,

Thomas C. Roberts
Director
Legislative Analysis Division

Enclosure

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RESPONSE TO QUESTIONS FROM CONGRESSMAN EMERSON

QUESTION 1:

It seems to me that the attitude of some at EPA is "there is no place for bad science, except for a good cause -- and we'll decide for the American people what a good cause is." Do you agree with that attitude?

RESPONSE 1:

No. We do not agree. This is not the attitude of anyone at EPA that we know, and it is not our attitude. The ETS risk assessment is well documented and responsive to the results of extensive scientific peer review. The anonymous quote cited in your question is in error. The risk assessment is good science, not "bad science."

QUESTION 2:

According to the news report, the ETS risk assessment was based on just 11 U.S. studies of ETS and lung cancer, and that only one of these studies produced a statistically significant result. The report says further that EPA could reach the conclusion that ETS is a Group A carcinogen only by combining these studies through a procedure called meta analysis and then lowering the statistical standard for evaluating scientific results from 95 percent to 90 percent.

RESPONSE 2:

The news report is in error. The conclusion that ETS is a Group A carcinogen is based on the total weight of evidence, specifically:

- the carcinogenicity of mainstream smoke from active smoking, with no evidence of a dose-response threshold;
- the chemical similarities of mainstream smoke and ETS, both of which contain over 40 compounds known or suggested to be carcinogenic to humans;
- supporting evidence from animal bioassays and genotoxicity studies;
- evidence of ETS exposure and uptake by nonsmokers; and
- data from 30 epidemiology studies of ETS and lung cancer from 8 different countries.

The only time the U.S. studies alone (the 11 epidemiology studies plus several exposure studies) were used was for the quantitative evaluation of the U.S. population risk. Once the lung carcinogenicity of ETS was established based on

the total weight of evidence, it was felt that the U.S. studies provided the best data for quantitatively estimating relative exposure levels and population risks in this country.

For the hazard identification assessment of the epidemiology studies of ETS and lung cancer, all 30 were analyzed a variety of ways, and each analysis demonstrated an association between ETS and lung cancer. The meta-analysis was only one of the analytical techniques that was used, and the overall conclusions are not dependent on the results of the meta-analysis. The meta-analysis was used as an objective tool, since it provides a means of including both positive and non-positive study results into an analysis. Furthermore, the meta-analysis procedure was specifically reviewed and approved by the EPA Science Advisory Board (SAB) as appropriate for analyzing the ETS database.

QUESTION 2A:

Do you think the EPA's use of a 90% confidence interval as opposed to the standard 95% confidence interval was justified?

RESPONSE 2A:

The criterion used to assess statistical significance for the ETS epidemiology studies was 95% significance for a one-tailed test. The 90% confidence interval corresponds to this test, whereas a 95% confidence interval corresponds with the 95% significance level for a two-tailed test. In statistics, a one-tailed test is warranted when one has *a priori* evidence that if there is an effect, it will be in a specific direction. In the case of ETS, this would be evidence that ETS exposure would cause adverse effects rather than being protective. The evidence that active smoking causes lung cancer, in a dose-response manner down to low doses with no evidence of a threshold, coupled with the commonality of chemical constituents of mainstream smoke and ETS gave us such *a priori* evidence.

Furthermore, the EPA's conclusions are not dependent on this criterion. For example, the exposure-response data for ETS are revealing: Ten of the 14 studies with sufficient data for a statistical test of quantitative association of dose and response show statistically significant exposure-response relationships; the probability of this occurring by chance is less than one in a billion ($p \leq 0.000000001$).

QUESTION 2B:

Are you aware of any other EPA risk assessments, or any risk assessment you have been involved with in the past, in which the standard was lowered from 95% to 90%?

RESPONSE 2B:

The 95% significance level for the one-tailed test ($p \leq 0.05$) with a 90% confidence interval is analogous to the 95% upper confidence limits for a two-tailed test typically used by the EPA for extrapolation from high to low doses when quantitatively estimating human cancer risks from animal data.

Epidemiologic data are generally assessed using 95% confidence intervals for a two-tailed test; however, the ETS database is unique in several ways. First, there is the *a priori* knowledge of the lung cancer effects of active smoking. Second, the ETS studies examine the effects of exposure to actual environmental levels of ETS rather than high occupational or therapeutic exposures to a chemical. Third, virtually everyone is exposed to some level of ETS; therefore, these studies are especially prone to exposure misclassification, which decreases the ability to observe a true effect.

QUESTION 2C:

Have you ever conducted research and reported results based on 90 rather than 95 percent confidence intervals?

RESPONSE 2C:

See response to 2B.

QUESTION 2D:

Do you know why the first draft of the EPA report applied 95% confidence intervals? Why did the individual studies that EPA evaluated use this standard as well? Why did EPA depart from this pattern?

RESPONSE 2D:

The first draft used the same one-tailed test ($p \leq 0.05$) as the second draft and the final report. In other words, the criterion for statistical significance was consistent throughout the development of the risk assessment. In the first draft, 95% confidence intervals were inappropriately presented in addition to the significance values for the one-tailed test. In the subsequent reports, the 90% confidence intervals were used to be consistent with the significance values for the one-tailed test.

The individual authors of the ETS studies themselves did not all use 95% confidence intervals. Four used 90% confidence intervals, 18 used 95%

confidence intervals (two-tailed test) and eight reported no confidence intervals at all.

QUESTION 2E:

Do you consider that it is convincing in a scientific sense to base risk conclusions on just 11 studies, none of which is statistically significant based on generally accepted scientific standards for assessment of data?

RESPONSE 2E:

As discussed above, the EPA conclusion of ETS as a Group A carcinogen is not based "on just 11 studies." It is based on the results of several hundred studies, including but not limited to 30 epidemiology studies on ETS and lung cancer from eight different countries.

Twenty-four of the 30 studies found an increased risk, and 9 of these were statistically significant. This proportion of statistically significant positive studies is highly unlikely to occur by chance (probability < 1 in 10,000). In addition, many of the studies were small and had a low statistical power to detect an effect at environmental exposure levels, especially since the study results are based on whether or not the spouse smokes, which is a crude measure of ETS exposure. A better indicator of exposure is provided by the studies that collected data on exposure level, i.e., on the amount the spouse smokes. All 17 studies with exposure level data found an increased risk in the highest exposure group; 9 of these were statistically significant, despite most having small sample size. This proportion of statistically significant studies is extremely unlikely to occur by chance (probability < 1 in 10 million). Even more revealing, 10 out of the 14 studies with sufficient data for a test for exposure-response association show statistically significant exposure-response relationships; the probability of this occurring by chance is less than one in a billion.

QUESTION 2F:

I'm told that EPA recently evaluated the data on the health risks of exposure to electromagnetic radiation. I understand the EPA found that 25 of 40 epidemiologic studies reported a statistically significant increased risk of disease with exposure to electromagnetic radiation, and yet called the results "far from conclusive."

You may not be an expert in the field of electromagnetic radiation, but as a scientist, do you consider it odd that EPA could reach no conclusions on the basis of the 25 statistically significant studies in this case but could do so in the case of ETS, where none of the U.S. studies is statistically significant overall?

RESPONSE 2F:

There is no inconsistency when the entire weight of evidence is assessed in each case as EPA risk assessment procedures require. Please note that the draft report (EPA/600/6-90/0005B) on the Evaluation of the Potential Carcinogenicity of Electromagnetic Fields (EMF) is still a draft report and does not represent Agency findings.

Many differences exist in the evidence available on ETS and EMF as described in the respective EPA reports. One of the major differences is that studies on the chemical similarities of mainstream smoke (inhaled during active smoking) and ETS and on the toxicological properties of mainstream smoke and ETS, provide a strong addition to the weight of evidence for the conclusions on ETS. Moreover, both mainstream smoke and ETS contain over 40 compounds known or suspected to be human carcinogens to which the lung is exposed, and for which biological mechanisms of action have been described. For EMF, these elements of a total weight of evidence are absent or uncertain as yet. Furthermore, the field frequency and, thus, the exposure that might cause an effect is uncertain.

QUESTION 2G:

Would you as a scientist feel comfortable concluding a health hazard from epidemiologic reports of a very weak elevation of risk, in the range of 2.0 or less?

ANSWER 2G:

We would feel uncomfortable concluding a health hazard from one or a very few epidemiologic reports with observed risk elevations less than 2.0 in most circumstances. However, consider that with epidemiologic studies of ETS and lung cancer, everyone is exposed to ETS; some of the "controls" or "never exposed" are actually exposed more than the "ever exposed." Thus, any risks observed comparing "ever" vs. "never" exposed will be lower than the true risks.

One way to try to control for this "exposure misclassification" bias is to compare the "most exposed" group in each study with the controls. Of the 17 (out of 30) studies which classified groups by level of exposure, all seventeen reported that the most exposed group had an increased risk, and nine of these comparisons were statistically significant. The chance that 9 out of 17 comparisons are statistically significant if there is no effect is less than one in 10 million. Thus, with ETS and lung cancer we feel comfortable concluding a health risk.

QUESTION 2H:

Are you aware of any substance EPA has classified as a Group A carcinogen based on only one statistically significant epidemiologic study? What about where there are no such studies?

RESPONSE 2H:

As discussed above, EPA classifies carcinogens based on the total weight of the available evidence, not on single studies. In fact, several substances have been classified as "Group A" carcinogens in the absence of case-control or cohort epidemiology studies on the specific substance. Diethylstilbestrol, for example, was classified in Group A on the basis of case reports. 2-Naphthylamine was similarly classified in Group A primarily on the basis of case reports. The benzidine-based dyes, Direct Black 38, Direct Blue 6, and Direct Brown 95, have been classified in Group A in the absence of epidemiologic studies on the individual mixtures, based on the fact that benzidine, a known human bladder carcinogen, appears as a metabolite in the urine of exposed workers.

Note that ETS is the only carcinogen in Group A for which the cancer risk in humans was observed at environmental exposure levels, rather than at occupational or pharmaceutical levels.

QUESTION 2I:

Do you think EPA should have included in its analysis all of the relevant epidemiologic studies on ETS and lung cancer, including the large and important one published last November, even if the result called the risk assessment's conclusions into question?

RESPONSE 2I:

Yes, if and when new evidence can affect EPA's conclusions, it is appropriate for EPA to consider it. In the case of ETS and lung cancer, 5 new studies have been published since the literature review cutoff data for inclusion into the analysis. Four of these, including the Brownson et al study published last November are already discussed in the Addendum included in the risk assessment report. As discussed in the Addendum, all 4 studies show positive associations between ETS and lung cancer and are very consistent with the report's conclusions. A fifth study on ETS and lung cancer by Liu et al (Am. J. Epidemiology, 1993; 137:145-54) was very recently published. In it the authors conclude "... among nonsmoking women, exposure to tobacco smoke from their spouses was also

associated with an increased risk (of lung cancer)." Thus, all 5 studies on ETS and lung cancer published after the report's literature review cutoff data support the conclusions of the EPA report.

Mr. TRAFICANT. Our next panel consists of the General Services Administration, the Acting Commissioner of the Public Buildings Service, Mr. P. Gerald Thacker, and the Acting Director of the Office of Personnel Management, Ms. Patricia W. Lattimore. And I ask that you—if you could summarize your report and with unanimous consent that their reports be submitted upon the record. Without objection, so ordered.

TESTIMONY OF P. GERALD THACKER, ACTING COMMISSIONER, PUBLIC BUILDINGS SERVICE, GENERAL SERVICES ADMINISTRATION; AND PATRICIA W. LATTIMORE, ACTING DIRECTOR, OFFICE OF PERSONNEL MANAGEMENT

Mr. TRAFICANT. And I don't know what the panel start up is. If you worked your own out, let me know. If not, we will let the lady go first. How is that?

Ms. LATTIMORE. Thank you, Mr. Chairman.

With your permission, I was going to ask to provide a brief summary and then just submit the full statement for the record.

I would just like to say that the Office of Personnel Management does welcome the subcommittee's interest in protecting the health of Federal employees, as demonstrated by this hearing. As part of our mission at OPM, we do engage in providing policy guidelines and technical assistance in agencies interested in establishing preventive health services programs.

In 1986, we initially sponsored a conference on smoking cessation here in Washington designed to show agencies how to establish educational programs about the effects of smoking and how to encourage their employees to stop smoking.

Two years ago, OPM required fee-for-service plans participating in the Federal Employees Health Benefits program to provide smoking cessation assistance. This requirement reaches the bulk of our work force, since 72 percent of our Federal employees are covered by fee-for-service plans in FEHB. Health maintenance organizations, though not required to do so, usually provide their own smoking cessation assistance services.

The Federal Government, as an employer, has a clear responsibility to protect nonsmokers from this undeniably serious health hazard in the workplace, and the Office of Personnel Management is committed to meeting this responsibility.

We strongly support the objectives of legislation to protect nonsmokers in Federal buildings. We defer to GSA and other interested agencies regarding the implementation of H.R. 881. I thank the subcommittee for the opportunity to address this issue. And I am available to answer any questions.

Mr. TRAFICANT. Thank you.

Now Mr. Thacker.

Mr. THACKER. Yes. Mr. Chairman and members of the subcommittee, we appreciate the opportunity to comment today on this bill.

The General Services Administration's Public Buildings Service provides work space for more than one million Federal civilian employees throughout the country. And the objective for the General Services Administration is to provide a productive, safe, and

healthy workplace for those employees and for the citizens who visit the public buildings under our jurisdiction.

In summary, Mr. Chairman, we support the intent of this bill to ban smoking in Federal buildings. As you are aware from a previous hearing, the current regulations issued by the General Services Administration prohibit smoking in Federal buildings except in areas specifically designated by heads of departments and agencies for smoking.

I think, as you have very clearly seen on the record today, in the testimony, the classification of secondhand smoke as a carcinogen has raised the question of how effective that current policy is. In most modern buildings, 75 to 85 percent of the air is recirculated. It is quite apparent that, even if there are separately designated areas for smoking, unless there are separately ventilated areas, that secondhand smoke is likely to migrate throughout the building.

So we believe that a ban on smoking may be the most cost-effective way to protect nonsmokers from this newly classified Class A carcinogen.

We do have, if we might be allowed to comment, at least a couple of observations about specific provisions of the bill.

Section 3(b) would require the General Services Administration administrator to issue regulations and to take actions that may be necessary to institute and enforce the prohibition within the executive branch. We suggest that you might consider it more appropriate for such authorities to be placed elsewhere, since we in the General Services Administration are not responsible for all of the housing for all employees in the Federal Government. And, in fact, Section 19, Occupational Safety and Health Act and Executive Order 12196 require each executive agency head to insure its own Federal workers' safety and health.

Therefore, we believe that Section 3(b) should be modified to require all agencies to issue implementing regulations. But it is a suggestion we hope you might consider.

Again, in summary, we support the intent of this bill to ban smoking in Federal buildings.

Mr. TRAFICANT. Thank you, Mr. Thacker, and I want to thank you personally for your expeditious response to our committee's request for the surveys that you performed within GSA. We thank you very much.

I would defer to Mr. Clyburn, first, for questioning, and I will reserve my questions after the new Member.

Mr. CLYBURN. Thank you, Mr. Chairman.

I think I remember Mr. Thacker's testimony back in February. Thanks so much for returning and to you, Ms. Lattimore, for being here.

Let me ask both of you—I don't know if I caught the essence of your last comment, Mr. Thacker. It seemed to be an acknowledgment of the previous or the current policy of leaving the implementation of the 1986 regulation out in the field at the local agencies. Do you mean to do that?

Mr. THACKER. Yes. If I may clarify my comment just a bit.

The current provisions of the bill would place on the Administrator of General Services the responsibility of enforcing all aspects

of the legislation. We would like to suggest that, since the General Services Administration does not control all of the space that is used by Federal employees, that it might be more appropriate to consider the present structure of enforcement of occupational safety and health legislation. That places clearly on the head of each department and agency the responsibility for providing a safe and healthy workplace for the employees within that department and agency.

So it is simply to recognize that the General Services Administration does not normally provide enforcement of this type on such a broad scale. Certainly, within the buildings that we control, we should enforce the legislation. But I think there is a mechanism already in place requiring the heads of departments and agencies to take this responsibility for the employees under their jurisdiction. It is simply a matter of questioning the enforcement authority this bill would give to GSA.

Mr. CLYBURN. Thank you. I understand.

Now, am I correct in understanding that it has undertaken a study of its own regarding this issue?

Mr. THACKER. I am not personally aware of that. That may very well be the case. R.J. Reynolds has undertaken a study.

Mr. CLYBURN. Are you aware of any other studies currently under way regarding this issue?

Mr. THACKER. That is of banning smoking in Federal buildings or——

Mr. CLYBURN. Of the effects of secondhand smoke.

Mr. THACKER. I personally am not, no, sir.

Mr. CLYBURN. Why then would you—tell me, you don't think the current policy is working? Is that what you are saying? Not effectively?

Mr. THACKER. Yes. Again, the current policy simply provides for a physical separation of smokers from nonsmokers.

With the finding that secondhand smoke is clearly a carcinogen, we have to recognize, that, in most buildings, a simple physical separation is not enough to insure that nonsmokers would not be exposed to secondhand smoke. Because of the recirculation of the air within most modern buildings, it is quite possible that, even though a nonsmoker is not physically in the same room with a smoker, that that nonsmoker would be exposed to secondhand smoke from the air being circulated throughout the building.

In that sense, then, if we recognize the secondhand smoke problem, there seems to be only a couple of options. One is, if we are to ensure that nonsmokers are not exposed at all, to provide for some separately ventilated areas that very clearly could not allow and would not allow the smoke to migrate into the building's circulation system. The second would be simply to ban smoking entirely within the Federal buildings.

I think the intent of this bill is to ban smoking entirely, and, certainly, that would be the most economical means of ensuring that nonsmokers were not exposed.

Mr. CLYBURN. That is unless a building, a new building being constructed, did, in fact, set up such a separately ventilated area.

Mr. THACKER. Yes.

Mr. CLYBURN. And you were about to occupy that.

Mr. THACKER. Yes.

Mr. CLYBURN. So then, if such were the case and it were to employ this new regulation, it would mean that, without regards to separately ventilated areas for those people who would want to smoke, it would still ban the building.

Mr. THACKER. Yes, that is my understanding of the bill as it is written now. Yes.

Mr. CLYBURN. Right. So what you are saying from your testimony then is that if there were language in this regulation providing for that possibility, then you would have no problems with it?

Mr. THACKER. I think the only question then would be the classical one, and that is who is going to pay for it.

Mr. CLYBURN. Now this is a brand new building that has already been paid for, and it is up, and you get a lease on the building, and you want to move in there.

Mr. THACKER. If we are leasing the building and the building has a separately venilated designated smoking area, then you are right. Then, likely, there would be no additional cost to the taxpayers to make use of that space.

On the other hand, if we are building a new Federal building and we are required to provide designated smoking areas that were separately ventilated from the rest of the building systems, there would be, I think, some additional costs to the taxpayers to construct that space.

And if we were required to provide such space in every newly constructed facility built by the Federal Government, I think those costs could be quite large. Our estimate of the cost to construct such a separately ventilated area in a newly constructed building, that is one where there weren't major modifications required to the existing system, might range as high as $50 a square foot over and above other costs.

So if the General Services Administration, for the portion of the public buildings for which we are responsible, were required to provide designated smoking areas in newly constructed buildings, I think the Congress should be aware of the cost of that potential and some provision clearly made for who is to pay for the cost of constructing that space.

Mr. CLYBURN. Thank you.

Mr. TRAFICANT. I thank the gentleman.

Perhaps a brief answer from both of you. As managers in the Federal Government do you support the uniform standard policy that would apply to all the same way?

Ms. LATTIMORE. OPM, yes.

Mr. THACKER. I think I would have to say, with some qualifications, yes.

If you would like for me to explain those, I think there are some departments, agencies and branches of government housed in General Services Administration space that may have some special considerations they would like to bring before the Congress. I could mention one.

In the case of the Federal courts, many of the judges are concerned about applying such a strict prohibition on smoking to jurors who come into Federal buildings under penalty of law and who may have to be in the building under some close supervision for ex-

tended periods of time. It may well be that some special consideration should be given to permitting departments and agencies to provide for separately ventilated smoking areas within a building under some conditions such as that.

But, in a general way, absolutely, yes, we do support prohibiting smoking in Federal buildings. That is the most economical way.

Mr. TRAFICANT. You really go out of your way to accommodate those judges, don't you, Mr. Thacker?

Mr. THACKER. Well, it is a very important branch of the government, yes, sir.

Mr. TRAFICANT. Yes, it is.

I thank both of you very much for your testimony and for your continued involvement as the Congress attempts to set a uniform policy. And any other information you have will be—the record will be open for your submissions. Thank you very much.

Mr. THACKER. Thank you.

Mr. TRAFICANT. Our next witness is Mr. Mark W. Hurwitz, the Executive Vice President, Building Owners and Managers Association International. He is from the private sector on this issue.

We are glad to see you Mr. Hurwitz.

TESTIMONY OF MARK W. HURWITZ, PH.D., EXECUTIVE VICE PRESIDENT, BUILDING OWNERS AND MANAGERS ASSOCIATION INTERNATIONAL (BOMA)

Mr. HURWITZ. Thank you, Mr. Chairman. I appreciate the opportunity to be with you today and to sit in and listen to the testimony that has taken place thus far. Hopefully, what I am able to provide will give another perspective on what you are confronting.

BOMA, first of all, is the Nation's largest and oldest trade association exclusively representing the office building industry. Our membership owns or manages over 5 billion square feet of commercial office space in the United States.

Of course, BOMA Members share your concern about secondhand smoke in Federal buildings. Most Americans spend the majority of their day indoors and building owners and managers have a responsibility to their tenants to provide and maintain a healthy indoor environment. If the Federal Government were to mandate a smoking ban in either public or private workplaces, BOMA would support this mandate as part of an overall effort to improve the quality of indoor air.

A healthy indoor environment is a marketplace concern, important in attracting and retaining tenants, including the Federal Government. Increased media and legislative attention, however, has caused this to become a higher profile issue in recent years. Reliable information is needed to address this situation effectively and responsibly. BOMA Members have long pushed for research on the sources of and contributors to indoor air quality problems, and we are adamant about the need for good guidance.

BOMA International has worked with industry groups and government agencies in developing our efforts on indoor air quality. We have also implemented a strong campaign to distribute sound guidance to improve the indoor air quality management programs in the commercial property community. In cooperation with the EPA—you may be interested in knowing—and our local associa-

tions throughout the country, which are in 87 cities throughout the United States, we have held a nationwide series of over 50 seminars based on the EPA manual Building Air Quality, which I know you are all familiar with, and have attracted thousands of people who are responsible for literally billions of square feet of commercial office space to acquaint them with all that is known about how high levels of high-quality indoor air can be provided.

I am also pleased and proud to say that, in January of this year, the BOMA International Board of Governors, which includes a representative from every major city in the United States, unanimously passed a resolution to support a Federal ban on smoking in the workplace, which I request, incidentally, this resolution be included in the record. We see this resolution as a responsible and pragmatic step forward.

Let me mention, again, we didn't just say Federal office buildings, we said Federal ban on smoking in the workplace.

Mr. TRAFICANT. Would the gentleman yield for a minute? That report was—you came up with your own resolution and report?

Mr. HURWITZ. In January, our Board of Governors unanimously adopted a resolution that we would support the action being contemplated by this committee and also we would not oppose—rather would support a similar ban for smoking in all office buildings, not just Federal office buildings.

Mr. TRAFICANT. Without objection, that resolution will be included in the record.

Mr. HURWITZ. Thank you.

[The Resolution referred to follows:]

RESOLUTION BEFORE THE BOMA BOARD OF GOVERNORS

Whereas, indoor air quality has been the focus of increased media, legislative and regulatory attention for the past several years, with expectations that the intensity of that attention will significantly increase in the months and years ahead.

Whereas, indoor air quality, as a market place demand within the commercial real estate industry, requires no regulation, but requires much information to address this situation effectively and efficiently.

Whereas, BOMA members have long pushed for further research on the sources and causes of indoor air quality problems, and we are adamant about the need for good guidance. We have worked with industry groups, environmental organizations and government officials in coordinating our efforts. We have also demonstrated a strong campaign to develop and distribute sound guidance information to prepare and improve the indoor air quality management programs in the commercial property arena.

Whereas, many legislative and regulatory efforts are underway to dilute or redistribute the sources of indoor air contaminants.

Whereas, BOMA firmly believes that the most effective course of action is to prevent contaminants from being introduced to the workplace in the first place.

Whereas, tobacco smoke has been identified as a leading cause of indoor air contamination, and has generated a great deal of concern within the medical/health community.

Whereas, in light of the EPA's recent classification of second-hand tobacco smoke as a "Class A" carcinogen, BOMA is even more concerned with the consequences to the health and well-being of office tenants, their employees and guests who are exposed to second-hand tobacco smoke in the workplace.

Be It Therefore Resolved, that BOMA would support a federal ban on smoking in the workplace, as part of an overall effort to improve the indoor environment. We do, however, have several concerns with a federal ban—first and foremost among them is that the enforcement of any ban should not be the responsibility of the nation's building owners and managers. The second concern is that any liability incurred must not apply to the owners and managers of commercial, rentable real estate when smoking does occur after a ban is imposed.

Mr. HURWITZ. Previously, legislation has proposed to address indoor air quality problems, including secondhand smoke, by mandating ventilation standards to introduce more outside air into a building. While this may dilute or redistribute polluted air, this does not solve the problem by controlling its source. Since most buildings do not have separate ventilation systems for designated smoking areas, secondhand smoke is distributed into nonsmoking areas of the building, endangering the well-being of nonsmokers.

As a side note, I, myself, lost my wife to cancer, and she indeed was a nonsmoker.

Separately ventilated areas for smokers are the only way to ensure that secondhand smoke is not redistributed into other areas of the building. GSA has already testified that, in new buildings alone, the design and installation of separate ventilation systems would cost $30–50 a square foot.

I need not spend any time about the EPA action on classifying tobacco smoke. I will move this along so we can get to the Q and A section.

We do have several concerns with a Federal ban. First and foremost, that its enforcement, we don't believe, should be the responsibility of the nation's building owners and managers. The GSA's policy is that the head of each agency is responsible for implementing smoking policies.

In cases where a Federal agency leases space in a multi-tenant office building with a building-wide smoking ban, the building manager, should be responsible for policing the employees of that agency or the other tenants. But we believe it is the tenants' representatives who should hold ultimate responsibility.

This all ties into the liability question, sir, that you keep raising and, indeed, we are very concerned about.

Our second concern is, we can label liability. The liability to be incurred, we believe, should not apply to the owners or managers of commercial real estate who in every way possible attempt to enforce the ban on smoking in Federal office buildings or leased space, where every good step possible is put into place to control smoking in the end.

Someone sneaks to the men's room or the ladies' room to have a cigarette. The owner of that building or manager of that building is held liable for that situation. That is likely to happen. We will do all we can, of course, to educate tenants and have enforcement programs in place, but the liability question greatly concerns us.

So in conclusion, we believe a uniform Federal smoking ban would enable building managers to more effectively control the quality of the indoor air environment, through eliminating one of the most harmful sources of indoor air pollution. If a Federal ban is imposed, BOMA members will support it, and BOMA International will do everything we can to educate building owners, managers and tenants to effectively ban smoking in the workplace.

Thank you very much. And I welcome any questions you may have, sir.

Mr. TRAFICANT. Mr. Hurwitz, there is a question that pops up.

When government leases space in the private sector who owns this space and the government says there shall be—if it is a sepa-

rately ventilated room, how much is that going to cost you, if you know, or who is ultimately going to pay?

Mr. HURWITZ. Well, ultimately, if you work it all through, the taxpayer. The developer and owner of the office complex is interested in doing business with the Federal Government, and BOMA members are. In fact, we would like to see less Federal building construction and more leasing from the private sector, but that is another issue for another committee at another time.

I think if a developer or owner wants to do business with the Federal Government and lease space, they must comply with the Federal laws and regulations, as they already do. So the choice facing them would be, do they install these systems? And, of course, this is a pass-through and this will reflect in the square footage rental rates, and, ultimately, who is paying the rent but you and I and the other taxpayers of America.

Mr. TRAFICANT. Do you have any types of cost data or any breakdowns? I know it is rather early in the matter on this issue on costs for ventilated types of specialized spaces in smoking areas.

Mr. HURWITZ. Our preliminary data, sir, lies pretty much with what the GSA believes, from a low of $30 to a high of $50 per square foot plus the annual cost of maintaining proper ventilation.

Mr. TRAFICANT. I ask unanimous consent that that data be included in the record. If there are no objections, so ordered.

[The following was received Mr. Hurwitz:]

Representatives Traficant and Clyburn requested supplemental information on the cost of building and maintaining separately ventilated smoking rooms in office buildings. In our testimony, we concurred with an estimate provided to the Subcommittee at a hearing on February 23 by P. Gerald Thacker, Acting Commissioner, Public Buildings Service, General Services Administration, that it could range from $30–$50 per square foot.

In newly constructed buildings, installation of a separately ventilated smoking room is much simpler than retrofitting an existing building (which, in many cases, is not even possible). In a new office building, the most economical way to provide smoking areas would be to install them in the same location on each floor, stacked, as is done with restrooms. For the first smoking area in each building (based on a room 10′ by 10′), the costs would include construction of a vertical shaft, ductwork, and a large fan (usually located on the roof) to draw the air out of the building. Total cost would average about $10,000.

Once one room is installed, costs for all subsequent smoking areas, provided they are stacked, would be significantly less. Costs would include a small fan for each floor and the extra ductwork going to that particular floor, and would cost anywhere from $700–$1,000.

These costs estimates do not include costs of maintenance (filters, etc.) or other "hidden costs", such as increased energy usage.

Mr. TRAFICANT. Mr. Clyburn.

Mr. CLYBURN. Mr. Chairman, thank you.

I have basically two questions, I guess. First one is, of course, a bit facetious, but you do realize that, if such were the case, the cost of building the buildings would actually be paid for by smokers, wouldn't it?

Mr. HURWITZ. Say again, sir?

Mr. CLYBURN. I said, if we were to do what the Chairman just said, that is the added cost for building, paying for a separately ventilated building, would actually be paid for by smokers, wouldn't it?

Mr. HURWITZ. If only smokers were taxpayers.

Mr. CLYBURN. If they were included among the taxpayers.

Mr. HURWITZ. Yes, sir. They would bear part of the cost.

Mr. CLYBURN. So I was a little bit facetious.

I do want to ask you one question about separate buildings. I saw a building not long ago, a residential building, that had two heat pumps outside of it. And I asked what that meant, and they told me it was more economical to have two systems in the house than one because if you have a condominium, up and downstairs, it would be more economical.

In view of that, how do we get to $30 to $50 a square foot on top? I am assuming that is not what your testimony is saying here. I am assuming you are saying it costs $30 to $50 more per square foot?

Mr. HURWITZ. Yes, sir. First, let me point out I am not an engineer. I am a PhD in organizational development and an association executive. But from my visits to our members' buildings as recently as yesterday in Fort Lauderdale, the way office buildings function, they have more than one unit. They have two, three, four. So, depending upon what the outside temperature is and so on, they can adjust for maximum efficiency.

If, for example, there were only tenants of a law firm working on a Saturday or Sunday to prepare testimony to come before this committee, we don't have to heat or air condition the entire building. Only one of those could be used and so on. So they are all going through the same duct system. It is just a matter of energy conservation.

During the hot, difficult time of the summer or cold winter days, perhaps they will move up to 70, 80 percent of the total capacity, but in order to maintain efficient energy management systems in most large office buildings there are two or more units also.

Mr. CLYBURN. Absolutely. So if——

Mr. HURWITZ. But they are not separate. They are going through the same ventilation system.

Mr. CLYBURN. But aren't there some separate ones? If you were to close off an office condo, if the ducts were closed off in one area, you are telling me to go with a separate system would cost $30 to $50 per square foot more?

Mr. HURWITZ. They could do that for a floor and-in some very sophisticated building, we could do that for a floor or even part of a floor, but as to existing systems are not in place in a large majority—if not a hundred percent—of the office buildings where a 10 by 12 space or one small smokers' area could be properly and separately ventilated without going back through the entire system.

Mr. CLYBURN. The report that will go into the record will have some detailed architectural information as to how you got to this $30 to $50 per square foot more?

Mr. HURWITZ. Yes, sir. We will provide that.

Mr. CLYBURN. Thank you.

Mr. TRAFICANT. Thank you, Mr. Clyburn.

Mr. Hurwitz, do you smoke?

Mr. HURWITZ. No, sir, I do not.

Mr. TRAFICANT. Were you once a smoker?

Mr. HURWITZ. Yes, sir, I was.

Mr. TRAFICANT. How many years did you smoke?

Mr. HURWITZ. About 20 years.

Mr. TRAFICANT. I want to thank you from the private sector for being here, being candid and frank. We appreciate it. Any other information you and your organization has as time moves on on this legislation, we would welcome it with open arms. And we thank you for being here.

Mr. HURWITZ. Thank you, sir.

One private matter, if I can take off my hat in representing my association. I feel confident, sadly confident, that my personal smoking played a role in my wife's death. I don't say that proudly at all.

Mr. TRAFICANT. Well, I lost a mother due to respiratory complications. She was a heavy smoker.

Mr. HURWITZ. Thank you, sir.

Mr. TRAFICANT. Thank you for your testimony, Mr. Hurwitz.

The next panel: Dr. Gio Batta Gori, Director of the Health Policy Center, Bethesda, Maryland; Dr. Maurice LeVois, Principal Scientist, Environmental Health Resources, Tiburon, California; and Mr. Simon Turner, Technical Manager, Healthy Buildings International, Fairfax, Virginia.

TESTIMONY OF GIO BATTA GORI, DEPUTY DIRECTOR FOR CANCER CAUSE AND PREVENTION, NATIONAL CANCER INSTITUTE; MAURICE E. LEVOIS, PH.D., PRINCIPAL SCIENTIST, ENVIRONMENTAL HEALTH RESOURCES, TIBURON, CA; AND SIMON TURNER, DIRECTOR, HEALTHY BUILDINGS INTERNATIONAL

Mr. TRAFICANT. Gentlemen, in which order do you plan to go?

I would ask if you could do, as have the others, summarize your statements. Your full statement will be placed in the record. I ask unanimous consent. There being no objection, so ordered. And if there is no pecking order, Dr. Gori.

Mr. GORI. Thank you, Mr. Chairman, members of the committee.

I am Dr. Gio Gori. I am a toxicologist with training in epidemiology and broad interests in smoking and health, cancer causation and risk assessment. I am President of the International Society of Regulatory Toxicology and Pharmacology and Fellow of the Academy of the Toxicological Sciences. My full resume is attached to my written statement.

The Tobacco Institute asked me to express my concerns as a scientist about EPA's recent report on Environmental Tobacco Smoke. These views, of course, are my own and are not necessarily those of the Institute. I must say that, given the testimony today, I feel like a dragon in a room full of eager St. Georges.

Without recourse to unnecessary technical detail, I intend to point out why the EPA report is actually a scientific scandal.

The actual amount of ETS in air is minuscule. Keep in mind that fewer than two dozen of ETS components can be measured and that their concentrations are thousands to a million fold below the corresponding levels that are permitted in the workplace by the Occupational Safety and Health Administration.

The EPA report itself shows that, in the course of an entire year, the average nonsmoker may be exposed to less tar than what may be obtained by smoking one single cigarette.

EPA considered only studies of spousal exposure in the home and its review cannot be relevant to workplace exposures.

Also, for the ETS report, as we have heard before, the EPA changed its own rules. The spousal studies review did not show that ETS leads to significant lung cancer risk until EPA relaxed and doubled the margin of error it usually uses in determining significance.

Let me put it in practical terms. If your political pollster introduced a result with a plus or minus 5 percent margin of error, you may feel comfortable even in a close race, but if he doubled that margin to plus or minus 10 percent, you would look for another pollster.

Mr. TRAFICANT. Excuse me, Dr. Gori. I would like to give you an option. The Congress has now called for a series of votes. I would like to give you the option either to conclude your statement or to suspend now and come back in the beginning with you so it would be fresh with all three.

Mr. GORI. Fine.

Mr. TRAFICANT. I don't mean to rush you in any form, but there will be a lengthy vote and then a number of shorter votes, and we are now checking to see how much time that will take. So I want to give you a full opportunity to offer your testimony in with the other three.

Mr. GORI. I would be more than glad to wait until you come back, Mr. Chairman.

Mr. TRAFICANT. You would rather wait?

Mr. GORI. Sure.

Mr. TRAFICANT. With that, we will temporarily adjourn.

Do you want to summarize your statement or do you want to wait until we come back?

Mr. GORI. I am prepared to wait until you come back.

Mr. TRAFICANT. We don't have an exact time period when we can come back. We would like to reconvene in a half hour if we could, but if that time frame is a problem for us, we will be here as soon as possible after that. And we ask you not to leave, and we apologize.

Our hearing has gone a little longer than we expected, but we have the time, and we will be back. If we can be back here in half a hour, we will notify the staff and then we will reconvene upon their notice. I apologize for the interruption. Thank you.

Mr. GORI. Thank you.

[Recess.]

Mr. TRAFICANT. We will reconvene. I don't know how many Members are coming back. And I do not want to be accused of bringing you guys on at the end when no one was here and I will leave it up to you.

We are going to have another hearing and there may be other people that may wish to testify in opposition to some of the findings. And I will leave that opportunity for you then if you want to come back and you would be one of the lead panels that would have an opportunity to be seen and heard, perhaps by more people as others were.

So I don't mean—I know it costs you money to come here. I apologize for that. Or you can also summarize what you are doing

here and you can also send representatives back. I would like to leave that open on the record, extend you that courtesy. Mr. Gori, what do you say.

Mr. GORI. I think our remarks are not very long, and given the sparsity of seats in the committee here, we are not likely to have many questions. So probably you can dispose of us rather rapidly and we can even come back at the next time.

Mr. TRAFICANT. Well, I have questions for you, so if we keep you here, I want you to know I do have questions for you. So with that, we will go forward and reconvene. Dr. Gori.

Mr. GORI. I will pick up where I left last time, and I am glad that you will have a chance to be here and to listen to this. As I said, I will focus on the issue of why the EPA report is not scientifically sustainable.

The actual amount of ETS in air is minuscule. Keep in mind that fewer than two dozen compounds can be measured and their concentrations are thousands to a million-fold below the corresponding levels that are permitted in workplaces by the Occupational Safety and Health Administration.

The EPA report itself shows that in the course of an entire year, the average non-smoker may be exposed to less tar than what may be obtained by smoking one single cigarette. EPA considered only studies of spousal exposure in the home and its review, therefore, cannot be relevant to workplace exposures.

Also for the ETS report, as we have heard before, the EPA changed its own rules. Let me put it in more practical terms. If your political pollster produced results with plus or minus 5 percent margin of error, you may feel comfortable even in a close race, but if the margin of error was plus or minus 10 percent, you would look for another pollster.

This is precisely what EPA did in this case. Now, despite much epidemiological evidence to the contrary, EPA also decided to ignore several manifest lung cancer risks such as differences in nutrition, disease experience, physical activity, socioeconomic status, occupations, and others. All confounders that are more than capable of accounting for the small risk attributed to ETS by the agency.

EPA also decided not to consider 15 epidemiological studies of ETS in workplaces. And two recent and federally funded large studies which, together, void the agency's conclusion when added to the body of evidence. The EPA report of ETS may indeed be an effective policy instrument, but its claim of scientific support is absolutely misrepresented.

Surely EPA thinks tobacco control is a legitimate political issue. But does the end ever justify whatever means. As the national debate on scientific misconduct intensifies, should good intentions forgive an official report that otherwise would guarantee severe censure to any individual scientist or academic institution?

Mr. Chairman, members of the committee, these are no trivial questions in defense of tobacco interests. They obviously reach far beyond the ETS issue. Ultimately, their answers will determine the credibility of science and its continuing public support, and more important, whether civic institutions are indeed worthy of the public's trust.

Mr. Chairman, this concludes my statement. I am ready for your questions.

Mr. TRAFICANT. I thought perhaps we would go to Dr. LeVois and go through the panel and revisit. Is it LeVois?

Mr. LEVOIS. LeVois, and I am an epidemiologist. My office is in Mill Valley. I was asked by the Tobacco Institute to comment on the scientific issues pertaining specifically to the epidemiology that was relied on in the risk analysis. I have commented extensively for the record through the process and those comments are available to this committee if you are interested.

I, too, feel that it is probably unwarranted to rely on the EPA's risk analysis for the justification for ban of smoking in the workplace for a couple of reasons. And I realize from all that we have heard here today, that there are considerations that go far beyond the epidemiology, but that is all you really are focused on. So I would like to share with you my opinions of what is wrong with the science that underlies this risk analysis and just let that be part of the public record.

My primary concern is this: These studies are not really environmental tobacco smoke studies as several people before me have pointed out, including Dr. Farland. These are studies of the risk of getting cancer as the wife of a smoker compared to the risk of developing cancer as the wife of a nonsmoker.

What that means is that everything that goes along with the unhealthy lifestyle of a smoker—and it is well documented in the literature that they have unhealthy life styles and their spouses have similar duties, socioeconomic, status and exposure to risks on the job because of their socioeconomic status, alcohol consumption, local environment, right down to the House level is influenced by all of these things.

So what you have really is, you have a comparison of two groups that share very different types of risk factors for developing disease in general and lung cancer in particular. And to attribute the entire difference and risk—and it is very small—we are talking about without cranking in any of the adjustments that they use, about a 15 or 20 percent difference in these two groups.

That is such a small difference that, from the standpoint of epidemiology, without absolutely perfect study design, I would never feel comfortable. And I don't think in any other issue anyone in my field would feel comfortable interpreting such a weak design with such a small elevation or difference in risk. And, of course, these are not uniform. There are studies that showed no risks, showed a risk that is less in the wife of smokers than in the wives of the nonsmokers.

This kind of weak effect that moves up and down above and below any risk at all is a clear indication that using one-sided tests of significance, which are generally agreed in the medical literature never to be appropriate in biomedical research was an unwarranted relaxation of the standard of demonstration of a significant risk.

I have particular concern that the committee realize that there were 13 studies that included a look at the risks of ETS exposure in the workplace. And if you did the same sort of meta-analysis or pooling of the data of the 13 existing studies on the workplace ex-

posure, you came up with a risk elevation of essentially .01 or nothing.

So if you look at relevant literature to the workplace, you don't find an elevation in risk. And I believe that one of the reasons you don't is because you avoid in the workplace definition of exposure these spousal concordances confounding and by using factors, that in my view, not only undermine the interpretation of the spousal smoking, but would be certainly worth considering by this committee as workplace relevant data.

And I would second what Dr. Gori has pointed out, that the levels that we are talking about are extremely low. I am quite confident that there is smoking going on all over this building right now and so we are talking about eliminating this level of smoke contamination from buildings.

And I think it is important for the committee to realize that there are present in this room toxicants that are far more serious than that, the off-gassing of formaldehydes, of epoxides, of probably asbestos, of benzene, of benzo[a]pyrene, which is a ubiquitous product. It is undoubtedly being produced down in your kitchen right now.

And these levels are in the air and present at much higher levels from these natural sources and they are not just the result of smoking in these office spaces. And so if you just eliminate smoking, keep in mind that you are not eliminating any of these other toxicants that would be eliminated were you to implement better air filtering and air exchanges, better ventilation in the rooms.

So if the real purpose and underlying hope of this committee is to protect public health, you really are overlooking the obvious. Granted you can completely eliminate the smoke with a very simple ban, but I think that you are probably confusing real issues with matters of other policy relevance if you think that you are really protecting health.

Thank you.

Mr. TRAFICANT. Thank you, Doctor.

Mr. Turner.

Mr. TURNER. Yes, good afternoon. My name is Simon Turner. I am a technical manager and director of a company called Healthy Buildings International, which is a company that specializes in the study of indoor air quality. And we help property managers and architects of new buildings prevent indoor air quality problems in major buildings.

Just a few of the buildings we have looked at in the past, government buildings—I think these might be most relevant to you-include the Department of Health and Human Services, most of the Federal Reserve Banking System around this country which we currently do, many Architect of the Capitol buildings, the Longworth Congressional Office Building, the Supreme Court and the Library of Congress are some of the buildings that we have studied indoor air quality in.

In fact, over the last 12 years, we have looked at a total of 953 major buildings. I think what I want to go over is just some of the variety of indoor contaminants. For instance, in 33 percent of buildings we have looked at, we found fungi to be one the major indoor

air pollutants that you find growing inside the air conditioning systems.

And in decreasing order of magnitude, we find problems with dust, high humidity, bacteria, formaldehyde, fiberglass, car exhaust fumes, volatile organic compounds, tobacco smoke, high humidity, and ozone. In fact, our figure on tobacco smoke is 3 percent in these 950 buildings.

Bear in mind, too, that 20 percent of the buildings we have examined have been operating with no outside air, primarily to save energy. They are not designed that way. They are being operated with no outside air, which is recycling the same air round and round through the building and that is how you can begin to picture what a sick building might be.

When you are recycling that same air through dirty filters and perhaps dirty duct systems, that is often how the contaminants build up to levels where people get irritated. I want to make clear that the building managers that we are working with are working to correct those problems and prevent those problems in their building and working to educate their tenants to prevent these problems as well.

I want that to be understood, that we feel that property managers, at least those who we work with, are working on those things and that should be the focus of how we should try to encourage building managers to prevent indoor air quality problems.

We are talking about environmental tobacco smoke. And even if we assume for now that it is in fact a carcinogen, let's bear in mind that the EPA, who made this decision, have no authority to regulate in this issue. It is up to OSHA and other groups, other governmental groups to regulate ETS and other indoor and workplace environmental components or constitients, if you like.

And both OSHA and NIOSH and also the American Conference of Governmental Industrial Hygiene has limits, maximum limits to which you can be exposed to day after day, without adverse health effects. That specifically is what ACGIH stated about their limits which they call threshold limit values.

Now, we find whether we are talking about smoking or nonsmoking offices or homes, levels up to a thousand times lower than those standards that are set by OSHA and NIOSH and ACGIH. In fact, measurements we have taken show that there is more benzene which is one of those Class A carcinogens we have talked about in nonsmoking homes than there is in smoking offices.

And the reason is, there are so many sources of benzene apart from smoking. And in addition to that, we don't ventilate our homes. Generally we ventilate offices, but generally most homes don't get ventilation. And other studies, a review of the literature that was done by Holcomb, shows that the most benzene of all is found in transportation and then homes and then offices.

Let's look at another Class A carcinogen which is asbestos. And I know you asked a question about that this morning. The OSHA's limit for asbestos, their maximum limit to which we can be exposed to asbestos is 0.20 fibres per cc of air in a building. So if we take a 100,000 square-foot building, you can make some calculations about how many fibers you can be exposed to in that building. That means there are about 5.6 million fibers floating around in the air

of a 100,000 square-foot building. That still is acceptable as far as OSHA is concerned.

And what we are saying now, we have one molecule of tobacco smoke, we have to evacuate the building. And, to me, that doesn't make sense. We feel that the best, most logical approach to removing all indoor pollutants and making a comfortable, healthy work environment for tenants and occupants of buildings is what we call a building systems approach.

What that means is, you obviously start with the right design, you document that design, and then you apply it every day during that building's operations throughout the life of the building. And the ironic thing is, the standards are already in place to do that, to apply a building systems approach. It is just a question of education and using and applying those standards.

And if, in addition to that, if you still have complaints about tobacco smoke, there are other options apart from a complete ban which don't necessarily have to be expensive. And we have had a number of clients come to us and ask us about those options and we have been able to show them how they can accommodate smokers without resorting to a complete ban which they found to be particularly inconvenient. And I am happy to share some of those options with you if you want.

Finally, I just want to mention some research that we have done, or actually commissioned the Harris Research Organization to do, on people's perception of their work environment. This was done in 10 countries around the world, polling about 15,000 people about the perceptions of their air conditioned work environments.

What Harris polls found was that Australia and the United States share the lowest incidences of people smoking in their immediate workplaces. Anywhere between 15 and 18 percent of people still have smoking in their immediate surroundings.

And yet when you also ask about absenteeism as a result of symptoms of sick building syndrome, we find that Australia and the U.S. tend to share the other end of the scale, some of the highest incidences of absenteeism compared with other countries. So clearly just addressing the smoke issue is not addressing the issue of indoor air quality as a whole.

Finally, we are seeing a lot of so-called clean indoor air acts pop up all around this country, yet we still see sick buildings being evacuated, high absenteeism, school children getting sick from their schools, all in buildings where there is no smoking. The EPA's own headquarters building is a good example. That is one of the most notorious sick buildings in the world.

Let's address the cause of the indoor air quality problems we find, not just the symptoms, address ventilation, hygiene, and education. And I think that will add up to a much more cost-effective route to indoor air quality than just hanging up no smoking signs.

Thank you.

Mr. TRAFICANT. Your testimony was summarized, enlightening, well-presented. Before I ask my questions, I am going to defer to our Ranking Member, Mr. Duncan from Tennessee.

Mr. DUNCAN. Thank you, Mr. Chairman.

Mr. Turner, you said that the ETS problem is not the major factor in determining indoor air quality. What do you think, if we

were to try to do something to improve the air quality in buildings, what are the two or three most important things that can be done?

Mr. TURNER. Well, I think they probably would be. As I mentioned, first of all, make sure our buildings are properly ventilated. A good example of that is ASHRAE. The American Society of Heating, Refrigerating and Air Conditioning Engineers, a very well-respected group, set minimum ventilation rates for office buildings of 20 cubic feet per minute of outside air per person. And they say in that standard right there, in that standard, that this will accommodate a moderate amount of smoking.

So, obviously, we will want to encourage our building operators to operate them at 20 CFM. And that is good, most accept that. Good filtration. Get filters up to a minimum of 40 percent which is the ASHRAE dust spot test. And keep your system clean and prevent microbes from growing in the system. These three common sense approaches, I feel, will do most to eliminate indoor air quality problems in buildings and that is where we should be addressing the attention.

Mr. DUNCAN. Of indoor air quality problems, where would you rank tobacco smoke?

Mr. TURNER. Well, we have taken our database of nearly a thousand buildings and looked at that. And every time we identify an area of a building or one that has a significant problem with tobacco smoke, we mark it down as we do with any other indoor contaminants, and we find ETS to be about 3 percent.

Mr. DUNCAN. I heard you say that 3 percent awhile ago. What would be the percentage—what is the highest percentage?

Mr. TURNER. That is after all the buildings we looked at, 3 percent refers to the number of buildings out of that 953 that have problems that have been obviously connected with tobacco smoke.

Mr. DUNCAN. I see. All right. Dr. Gori, you said in your testimony, you say the EPA record is a glaring example of the misuse of science in support of preconceived policy aims. It is not unique, but it is possibly the most egregious example in a well-documented tradition of scientific abuse at EPA.

Are you saying, sir, that you believe the EPA had their mind made up before they ever started this study?

Mr. GORI. I think that the EPA itself in the report says exactly that. The report speaks clearly to this effect. The EPA said that they started with the preconceived idea that ETS was going to be a cause of cancer.

Mr. DUNCAN. I didn't get to hear your testimony, so why do you think that? Why do you think that this is the worst example of flawed science on the part of EPA?

Mr. GORI. Well, I don't remember seeing another report coming out of EPA with this accumulation of unscientific statements and unscientific practices.

Mr. DUNCAN. Okay. Dr. LeVois, you say in your testimony that EPA incorrectly attributes the entire spousal smoking lung cancer association solely to ETS. Are you saying, in this report, they didn't take into consideration any other possible factor or any other possible contributing cause?

Mr. LEVOIS. They didn't figure any other—well, let me put it this way: The studies themselves used and the risk analysis uses as its

primary focus the difference in the risk of lung cancer in the wife of a smoker compared to the risk of lung cancer in the wife of a nonsmoker, so that is the measure of ETS. It is not a direct measure.

As a matter of fact, in most of the studies, the wife was often deceased because lung cancer sometimes moves very quickly. A lot of the study's subjects were proxies. In other words, they were getting information from other people, not even the wife, about such things as exposure, her previous smoking, and so forth.

There was, in some of the studies, an adjustment made for certain things such as age. Sometimes they would adjust for one or two other factors. One of the things that is most disturbing to me about the literature, in general, is that as more and more criticism mounted directed at the methods of these studies and questioning whether or not this level of risk could really be interpreted as an ETS risk, a lot of examples—confounders—were introduced.

And the studies that came later don't address those, or if they did, they didn't publish the results. For instance the Fontham study that has been mentioned said, in its rationale for its own existence, I am sure that this was in its application for funding, that it was going to measure diet and occupation and a number of other biases and confounders.

But when the published report came out, none of those factors were addressed, only the very core, most minimal set of biases and confounding information was addressed. Stockwell is another important study which came recently. These studies have actually published or are intending to publish dietary effects independent of the smoking study.

And they published the smoking study independent of the dietary and work history and all the other effects, so you can't do the kind of adjustment for even the minimal set of biases and confounders that data were collected in many cases. And one way to explain that is this publish or perish syndrome when people are trying to milk a bulletin for as many studies as they can get.

But when the National Academy of Science report lists bulletin after bulletin of weaknesses in the design of studies that needs to be addressed and the application include the addressing of those weaknesses, and the publication doesn't show the adjustment, I think we are going too far in milking these for studies and ignoring the reason why we are collecting all the data.

Mr. DUNCAN. Neither of you are saying it is impossible to get lung cancer from secondhand smoke. You are just saying it is not scientifically proven?

Mr. LEVOIS. That is essentially right. I would never say it is impossible, because we don't know half of what we need to know about individuals' susceptibility and who knows where that can lead. Right now, given what we know, the fact is that good, sound epidemiology in the workplace from other settings on other chemicals clearly indicates that there is a point at which the zero threshold single molecule hypothesis simply is not realistic. The example I give you is that the air we are breathing is full of these molecules. You walk through them.

Mr. DUNCAN. Was it you that mentioned the benzene problem? Someone mentioned benzene. How much more likely are you-how much bigger a problem is that than the secondhand tobacco smoke?

Mr. LEVOIS. One way that I could address that, I didn't want to go through this table, but benzene, if you took an office space that was 10 feet by 10 feet by 10 feet high, so a nice small cubicle office space, you would have to burn 6,250 cigarettes simultaneously to reach the benzene level that is the threshold limit value for occupational settings.

So there are people that work driving buses, pumping gas, work in the cafeteria downstairs——

Mr. DUNCAN. I will tell you, I was a judge before I came to Congress. I wasn't a rocket scientist or intellectual. I don't have the slightest idea of what you said. What kind of threshold was it?

Mr. LEVOIS. Okay. There is a threshold limit value. There are a number of agencies that set levels at which—OSHA is one—but there are others that set limits on permissible levels of exposure levels which, although you can do hypothetical dose response modeling, there are no thresholds in hypothetical dose response modelings.

But these agencies set levels that are permissible for exposure recognizing that we are exposed to those levels everywhere, every day.

Mr. DUNCAN. Where does the benzene—what produces the benzene? I am not really familiar with that.

Mr. LEVOIS. Benzene is practically ubiquitous. It is produced in the kitchen downstairs. It is produced—every time you pump gas, you are exposed to high levels of benzene that are measurable in your lungs for days, whenever you drive in a bus, any mechanic that works in a garage. These are levels that are not just the threshold limit value.

In some cases, they are seriously high values and can in fact represent, because they get so much higher than that, genuine health problems. But this threshold limit value is selected because, practically you can't get below it in very, very many common situations in daily life.

Mr. DUNCAN. Let me ask one last question. Mr. Turner in these 953 buildings that you have studied, have the building managers or owners, or the people that you deal with, have they started out with tobacco smoke being one of the main complaints or the main thing that is they want to get out of the buildings.

Mr. TURNER. Generally not. There are many different reasons why property managers ask us into their buildings, very often preventative, not because they have a specific problem. They want to diagnose those because they want to prevent indoor air quality problems. They will have us in on a six-month basis.

In a way, those are the property managers we should be congratulating because they are taking a preventative approach. They are not looking at one individual contaminant, they are looking at the whole picture. They are asking us to come and investigate the entire building and do everything we can to make sure their building and their air conditioning system is providing good quality air. And as I said earlier, to me, that is what we should be trying to encourage.

Mr. DUNCAN. I said that was going to be my last question, but let me ask you this: We have smoking and nonsmoking sections now in all the restaurants and so forth. Does that work or does the smoke filter over into the nonsmoking section? Has that type of thing been effective?

Mr. TURNER. There are obviously lots of options for designating smoking areas in buildings and we generally find, if there is a little bit of care put toward the way you do it, they are very effective. All you need to do is have a little understanding about how well mechanical systems of air conditioning system work.

And you can in fact separate smokers by using the air conditioning system in terms of negative and positive pressures to prevent nonsmokers being exposed to tobacco smoke or by setting up a designated smoking area that exhausts the air directly outside. And you can do that with the use of an exhaust fan and the toilet system in 49 of 50 States without breaking building codes and that works very effectively.

So there are really a lot of different options property managers and employers have if they want to separate smokers. And in a sense you can look at each one on a case-by-case basis to see which people would be most comfortable with.

Mr. DUNCAN. Thank you very much.

Mr. TRAFICANT. Mr. Turner, there are many contaminants in the indoor recirculated air closed system, correct?

Mr. TURNER. Yes.

Mr. TRAFICANT. To the best of the your knowledge, are there any synergistic effects that are compounded by EST in conjunction with these other chemicals you have alluded to?

Mr. TURNER. We don't know a lot about that at all. I am not sure we know anything about synergistic effects. I have seen some research on the combined effect of radon and ETS. My recollection of it, although I don't pretend to be an expert on the radon issue in particular, is that with ETS particles when they combine with radon, they actually lower the dose of radon, ironically. I don't believe there is a lot of evidence about synergism at all. I haven't seen much more on that.

Mr. TRAFICANT. Are you a scientist?

Mr. TURNER. I like to think so. I have investigated air pollution and atmospheric chemistry since I left college.

Mr. TRAFICANT. Dr. LeVois, do you consider smoking the cause of lung cancer?

Mr. LEVOIS. I think that that is a very important question, because there is such a lot that has been said today about smoking, and I don't want to imply that I am an advocate of smoking. I actually tell everyone that I work for that smoking is the most important risk factor for lung cancer at all.

But bringing up smoking as it was brought up today, repeatedly, I think should underscore the point that a couple of people incorrectly made, that you can do dose symmetry from the act of smoking risks and come anywhere close to the risk that we are seeing reported in the epidemiologic studies of spousal exposure that the EPA is relying on. In fact, if you take the dosimetric modeling that is a standard model that the EPA uses throughout all of its other risk assessments, you come up with levels at the ETS dose range

that are thousands of times lower than what is showing up in these spousal smoking studies.

Mr. TRAFICANT. Dr. LeVois, I wasn't asking you about ETS. Do you consider smoking tobacco a cause for lung cancer?

Mr. LEVOIS. I think I have already answered that question.

Mr. TRAFICANT. What is it?

Mr. LEVOIS. I believe that it is the most important risk factor that has been identified.

Mr. TRAFICANT. That is to say, to interpret your language, yes?

Mr. LEVOIS. It is certainly the most important risk factor.

Mr. TRAFICANT. But you would not consider secondhand smoke a significant risk factor; is that your testimony?

Mr. LEVOIS. Not a significant risk factor. It is a theoretical possibility that hasn't been ruled out, but I really don't believe that the data that we see from these spousal smoking studies can be relied on because they don't rule out bias and confounding.

Mr. TRAFICANT. As an executive that would be responsible for the control of a workplace, and from the nature of your managerial duties, given the widespread documentation of—to use your terms—concern over ETS, would you consider yourself to be liability free to any litigation that may be brought against you by an individual that claims this secondhand smoke is causing them a problem?

Mr. LEVOIS. I am not an attorney and I have never served as an expert witness in a litigation situation.

Mr. TRAFICANT. It is not a legal question. It is not a legal question, it is a management question.

Mr. LEVOIS. My own position would be, if asked by attorneys do I think that there is reasonable standard of proof in a situation like that, would be no, I don't. I don't think that causation has been established and, therefore, I don't see how you could say that this individual has been harmed by the substance.

Mr. TRAFICANT. Is there a smoking policy in your company? Your organization?

Mr. LEVOIS. There doesn't have to be. There isn't a single smoker in my company.

Mr. TRAFICANT. There is not a single smoker.

Mr. LEVOIS. No. No one smokes. Hardly anyone does out in Mill Valley, California.

Mr. TRAFICANT. You made a statement earlier that smokers are not model health studies for such assessment, if I recall some of your language. You said there are many collateral issues involved here and we found that smokers are basically many times poor, don't have the same good nutritional habits and you classified smokers as being not as intelligent or not as health conscious, or would you say reckless or impervious to sound health and medical advice. You classify the smoker in a different light than anybody who testified before this committee.

Mr. LEVOIS. Well, I can tell you that the literature on the risk factors for lung cancer are replete with evidence that there is a correlation between all of the other risk factors that includes occupational exposure, mining lung disease. Lung disease in itself without regard to mining is a significant risk factor of lung cancer.

If you have a socioeconomic variable that is strongly related to cigarette smoking, you will also have linked in that pulmonary dis-

ease. You have all of the situations that lead, in the literature at least, to the developing of lung cancer, and there is a constellation and smoking behavior is at the center of it.

Now, I am not trying to malign smokers, but there is ample evidence to believe that these risk factors for lung cancer don't come in isolation.

Mr. TRAFICANT. Then can one assume, then, that if you take the smoking away from the smoker, as you have portrayed this smoker here, identified this smoker, that the smoker would still probably develop lung cancer; is that your testimony?

Mr. LEVOIS. Well, I wouldn't say that at all because it is such an important risk factor. So I think if you took the smoking away, you would reduce the risk, but you would still have an excess risk. With the same remaining risk factors, you would have an excess risk in that group.

Mr. TRAFICANT. I think one statement we can make is that God knows, unless we are atheists, we believe, so they know that not everybody knows everybody, unless we personally have knowledge. So if someone says the wolf is outside, who knows, unless we have seen the wolf.

If five people say the wolf is outside, you send your child outside, I mean at what point do you go beyond that fine line with the presentation of documented evidence that is not refuted in the scientific circles except for those organizations that have either contracted with, have been spokesmen for or have represented to some degree the interests of the tobacco industry?

Mr. LEVOIS. Actually, I don't believe that is true. The statement by Dr. Feinstein stands as an example of that.

Mr. TRAFICANT. We will have Dr. Feinstein in. We will have him speak for himself.

Mr. LEVOIS. You also ought to ask Drs. Winder and Cabot. Dr. Winder, at the American Health Foundation, has expressed serious reservations and he has been doing lung cancer epidemiology for 20 years. He has probably looked at more lung cancer patients and done more epidemiological research than anyone in this country.

He notes that the active smoker exposes themselves to very high degrees of heat and astringent properties, and these are promotional in and of themselves. You do not see these kinds of precursors and these kinds of collateral exposures that would lead him to believe that this is a causative association. It is not just the three of us sitting here that have expressed doubt that this body of epidemiologic evidence proves anything.

Mr. TRAFICANT. But you are expressing doubt.

Mr. LEVOIS. Yes.

Mr. TRAFICANT. The Congress of the United States has a report by the Environmental Protection Agency where they have been advised that there is no doubt in their mind, there is no doubt in the Surgeon General's mind, and we are not here about doubt. We are here about their reports.

Are you factually telling this committee that the Surgeon General of the United States of America, the Environmental Protection Agency, and all of these other awesomely large number of reports from many different scientific entities and communities are wrong

and false and we should treat them as such, with doubt? Is that what—basically what your testimony is? Yes or no.

Mr. LEVOIS. The Surgeon General this morning said the information was flawless at one point. She said the exposure is comparable to smoking. I doubt that also. My response to you is, I am giving you the benefit of my views on the problems of an epidemiologic argument that is flawed.

Now, if you don't see it as flawed, and if others don't see it as flawed, that is part of the scientific debate. But there are problems with that design.

Mr. TRAFICANT. Do you agree that these other groups do not present it as a guesstimate and doubt? They present it as an outright warning to the Congress of the United States? Do you agree with that?

Mr. LEVOIS. Actually, there were margins of error that were presented with all of these reports.

Mr. TRAFICANT. Doctor——

Mr. LEVOIS. You heard one thing, I heard another.

Mr. TRAFICANT. Doctor, I want to ask you a question. Are you testifying before this committee that the Environmental Protection Agency and the Surgeon General has presented findings that they believe could be doubtful? Or in other words, are you now saying that the Environmental Protection Agency and the Surgeon General of the United States of America are not really warning us, they are just going through an exercise with us?

Mr. LEVOIS. I think the warnings are there. That is not what I thought you were asking. I thought if there was uncertainty to those warnings, I think that you should ask possibly Dr. Baird to speak for the EPA or someone to speak for the Health and Human Services. But I believe that there is uncertainty. In my view, the uncertainty is rather important.

Mr. TRAFICANT. What state are you from, Doctor?

Mr. LEVOIS. California.

Mr. TRAFICANT. Have you testified before any commissions in the State.

Mr. LEVOIS. No, I haven't.

Mr. TRAFICANT. Isn't it a fact that the governor has banned smoking in government buildings?

Mr. LEVOIS. I believe that is right.

Mr. TRAFICANT. Isn't it a fact there are many other States that have banned smoking?

Mr. LEVOIS. I will take your word for that. I don't follow that.

Mr. TRAFICANT. As a professional and seeing the preponderance of the evidence that is being accumulated and presented to lawmaking authorities, do you believe it is imprudent for the acts that these policy-makers have taken?

Mr. LEVOIS. I don't think that will protect people from developing lung cancer. At least it won't protect the nonsmokers exposed to the kinds of levels that we are talking about here. It may be symbolically prudent, but if you are asking do I think that there is a real risk that is measurable in these studies, I don't think so.

Mr. TRAFICANT. Dr. Gori. Is it Gori?

Mr. GORI. Yes.

Mr. TRAFICANT. Do you believe that the inhaling of tobacco smoke in the lungs on a regular basis causes lung cancer?

Mr. GORI. It is a major risk factor for lung cancer. No question about it.

Mr. TRAFICANT. And if a person worked in a Federal building limited by space and was confined to one of the small cubbyholes that sometimes public workers—Federal workers who are often trod upon are relegated to, they were there with a chain smoker, would you feel comfortable telling this committee, in light of recent scientific data, that you would not be concerned for their welfare?

Mr. GORI. I have no way of judging your hypothetical, Mr. Chairman. In general, I would think that with the technological achievements that we have today, the capacity that we have to ensure good quality of air in a building, I would make sure that the recommendations that, for instance, Mr. Turner here suggested are in fact put in place.

Mr. TRAFICANT. But you do feel at least a need to clean up the quality of the trapped indoor air; is that what you are saying?

Mr. GORI. Provided that we understand that the sources of contamination in any environment, including this one here today, is not only environmental tobacco smoke. As we have heard, environmental tobacco smoke may be a minor contributor to the contaminants that circulate in the air that we breathe.

So it looks to me that if we really are concerned about the general health of people in any building, we have to look at the picture globally rather than attacking any particular substance by itself.

Mr. TRAFICANT. If you could just strike that magic wand and remove all benzene from a building, would you do it?

Mr. GORI. You are again putting a hypothetical that is not possible.

Mr. TRAFICANT. I am asking if you could, would you do it. Benzene is classified as a Class A carcinogen.

Mr. GORI. I could do so many things, Mr. Chairman, if I had this magic capability. All I am telling you is that you will not. You will not be able to eliminate benzene from any air quality or from any contact with humans. The EPA, 10 years ago or so, put out a pamphlet which unfortunately I don't have here with me that shows, for instance, that every person is exposed to over a milligram of benzene daily from natural sources, including eating a couple of eggs. A couple of eggs contains almost half a milligram of benzene.

Mr. TRAFICANT. It would be very difficult to just snap your finger and get rid of benzene?

Mr. GORI. It would be impossible, Mr. Chairman.

Mr. TRAFICANT. Do you believe that Congress can remove smoke from the workplace?

Mr. GORI. Sure, Congress has many ways that it can act and remove not only smoke but whatever Congress likes from the environment.

Mr. TRAFICANT. You just made a statement that it would be very difficult to remove benzene and we will take that answer and accept it from our hypothetical model here. But it would not be quite as difficult for Congress to remove tobacco smoke.

Mr. GORI. No, it would be very simple, I am sure. The point is, you would not be removing much from the contamination of your

building, and so it would be a token gesture rather than a real assurance that the air is going to be clean and good quality.

Mr. TRAFICANT. In the context of toxicology, as the distinguished gentleman from Tennessee mentioned, you know, we are not all scientists and we are just trying to do a job. But the toxicologists do the classifying of these respective substances. And do you challenge the EPA's classification of ETS as a Class A carcinogen?

Mr. GORI. Of course I do, Mr. Chairman. I have been challenging the EPA classification, not only on ETS, but on many other substances for 20 years. The fact is that most of the classifications that EPA comes up with are not based on science, but they are based on arbitrary administrative assumptions, which is written in several documents in the Federal Register, published there over the last 15 years.

Mr. TRAFICANT. So, would it be safe to say, then, that you are not all that concerned about asbestos in buildings as EPA is?

Mr. GORI. At the levels that we are talking about and with the program that the EPA has in mind of removing asbestos from building where asbestos is already present, I certainly am against that particular policy and I am not the only one in the country to say that removing asbestos from buildings, where asbestos is already in place, is going to cause more problems than leaving it there.

Mr. TRAFICANT. To the best of your knowledge, has there been any contention to the Class A carcinogen rating of asbestos in any lawsuit or is it a given, respected by the courts of law, as identifying asbestos in litigation and for litigation purposes as a Class A carcinogen without doubt, without question, without review, without appeal, to the best of your knowledge?

Mr. GORI. No. What I have to tell you, though, is that most cases involving asbestos and litigation have been in regard to workplace exposures to very high levels of asbestos for which there is quite clear epidemiological evidence of a correlation with mesothelioma or lung cancer if you wish. But here we are talking about doses that are far below what anybody would be exposed to in an occupational setting.

Mr. TRAFICANT. Given that answer, in a court of law, will a judge come back and go through all of these changes or will he look at ETS in a lawsuit as a Class A carcinogen or not? You are giving an opinion. I am trying to establish how the Congress and the taxpayers are going to face this issue. You have heard of workmen's compensation, have you not?

Mr. GORI. Yes.

Mr. TRAFICANT. You have recognized the fact that it has become one of the most highly escalating and most explosive cost factors for all the government; is that correct?

Mr. GORI. I have heard that, but I have no—I am not a lawyer, so I really don't know the details of this particular charge, Mr. Chairman.

Mr. TRAFICANT. Do you concur with Dr. LeVois' statement that smokers are basically not good health role models and are different from the nonsmoker or normal population?

Mr. GORI. I don't have to concur with that. There is plenty of literature showing that smoking households have a number of other risk factors that segregate specifically in these households.

Mr. TRAFICANT. Mr. Clyburn.

Mr. CLYBURN. Thank you, Mr. Chairman. I do have a couple of questions.

If I may build upon what seems to be a legal question, I know that I don't think any of you are attorneys. Just from your own experiences, if you were to look at the EPA study and were called upon in a court of law—in keeping with some of what we have heard today about the liability factors, if you were called upon in a court of law to give your professional opinion as to whether or not the EPA study stood on real good, solid legal foundation, I would like to hear from each of you as to what your reaction would be?

Mr. GORI. Mr. Clyburn, I again say I am not a lawyer as you perceived, but I would think that in my opinion, the EPA report would not stand scrutiny, close scrutiny in a court of law. I think that there is no doubt, and no one could deny that the EPA has been selective in using the literature that was supportive of their case and ignore the literature that was not supportive of their case.

This is not my opinion. It is a matter of record. It is a matter of fact, something that anybody can control and verify.

I just want to read you a passage here from a booklet "Good Science and Responsible Scientists." It was published by the American Association of Advancement of Science in conjunction with the American Bar Association and National Conference of Lawyers and Scientists. This is a booklet that summarizes the findings of this group and recommendations of this group as far as scientific misconduct is concerned.

One of the items that classifies as scientific misconduct is as follows:

Incomplete citation of previously published work or skewed selection of data to hide or disguise observations that do not fit the other's conclusions. I think on this basis alone, we should have enough evidence to question the results of the EPA report.

Mr. CLYBURN. Same question to you, Mr. LeVois?

Mr. LEVOIS. LeVois.

Mr. CLYBURN. I am sorry. There are some cultural differences here, my not being from California.

Mr. LEVOIS. I would have to tell a court of law, if I was asked to comment on that situation, exactly what I have told the EPA in extensive written comments and what I have told this committee. After reviewing the epidemiologic literature, which is the one area where I am an expert, I see serious problems in interpreting spousal smoking studies as if the only exposure were ETS and attributing all of that exposure or all of the very small excess risk to ETS.

It just would not stand up if it were anything but smoke. It is a very, very weak risk. You see in the literature reference to ratios of two to three of being the lower threshold in an increase in risk where epidemiologists feel reasonably confident in interpreting relationships.

We are talking about a relationship that has a risk that is one-tenth that bottom threshold. So you have to understand that while they are projecting thousands of lung cancers as a result of this very small risk, that doesn't change the epidemiology. The epidemiology is not very strong and interpretation of such a weak risk with a weak design is very uncertain.

Mr. TRAFICANT. Mr. Turner.

Mr. TURNER. From my point of view, I am neither a lawyer or an epidemiologist or a toxicologist like my panel members here, so I can't answer your question directly. But not wishing to avoid it, I will tell you what our role is in that when we work in buildings.

It is very difficult for us to dictate smoking policies, either to the property manager or the tenant, and instead, we have got to try to accommodate all of those activities that tenants and property managers want for them to happen in their building.

If they want to install some kind of smoking policy, we will help them do that. We will show them how to design a smoking lounge which will be effective. If they wanted to remove the carpets to get rid of organic emissions, some of which are Class A carcinogens, or remove office products—you can go on and on like that.

That is what we try to do with them is limit and reduce all of the pollutants at the same time with proper building operations.

Mr. CLYBURN. One of the buildings you have worked on is the Longworth Building?

Mr. TURNER. Awhile ago.

Mr. CLYBURN. You didn't work on the Cannon Office Building?

Mr. TURNER. No, I don't believe so. We have the Architect of the Capitol as one of our recent regular clients. In fact, they were talking to us yesterday about another environmental issue, but not that building.

Mr. CLYBURN. Mr. Chairman, if I may, you know I have never had any sinus problems in all of my 50 years until I moved into the Cannon Office Building. My eyes have been running, my nose has been running, the roof of my mouth has been dry. I don't know. Nobody in my office smokes. I wonder what's going on there now.

Mr. TRAFICANT. A lot of gas in that office building.

Mr. CLYBURN. I was wondering whether it might be a lot of hot air coming out of the hall.

I have got one other question I would like to ask in view of that. It was testified to earlier today, I don't know if you were in the room, but someone testified earlier that it would cost on the low side, $30, on the high side, $50 per square foot more if you were going to put up a new building and provide for separately ventilated smoking areas.

If you were here, you know I questioned that. In view of what you just said in your experiences and your background, would you agree with that?

Mr. TURNER. Well, I remember somebody asked a question, how did they come up with those figures.

Mr. CLYBURN. That was me.

Mr. TURNER. I, too, don't know how they came up with those figures. They may have been talking about installing a completely separate air handling unit in the ceiling void above the space of a designated smoking area. But you don't have to do that to have a

perfectly well-exhausted, designated smoking area that prevents any exposure of tobacco smoke to nonsmokers.

For instance, what you could do is just have a small exhaust fan there that hooks into the toilet exhaust system. And, again, I am not sure exactly what that would cost, but I am sure it would be a fraction of that kind of figure.

And if you are designing a building from scratch, you can do that and it just means routing the duct work in such a fashion and making sure the system is properly balanced. And every building is balanced as a matter of course when it is constructed in any case. The air conditioning system is balanced.

So it's hard for me to believe that, if you did it that way, which would be effective and perfectly in compliance with codes and standards that we have for ventilation, that it would cost that much. But, again, I don't know how they came up with that figure. I don't know exactly what costs would be involved until you looked into the specific building.

Mr. CLYBURN. I know we have got to go, Mr. Chairman. I would hope if we are going to have additional hearings, I am particularly interested in Mr. Turner, because I would hope that we are talking here, once again, as has been established this morning and agreed to by the illustrious Surgeon General, that we are here about protecting nonsmokers from smoke, not about violating the rights of smokers.

And so if that's what we are all about on this subcommittee, then I think Mr. Turner and his background, experience, and certainly from what I have seen in his testimony, is somebody we ought to have back here when we really get down to trying to make a decision on this.

Mr. TRAFICANT. Thank you. I would ask if we could expedite the answers a little bit. We are going to try to expedite the questions.

Mr. DUNCAN. Mr. Turner, just very briefly let me ask you: You know we are talking about banning smoking in Federal buildings and I am sure you have served in all of these 953 buildings—you have studied all kinds of different buildings. And I understand, in some buildings, you could open the windows. But then with the air conditioning and so forth, we started buildings that had windows you couldn't open.

The Capitol is a Federal building. It opened in the 1800s. We have got other buildings that opened in the seventies and eighties and nineties and so forth. We are really dealing, then, with a situation that varies from building to building, aren't we?

And where the smoking and nonsmoking sections might work in one building, that might not work in another building where the ventilation and so forth and the systems weren't quite as good; is that correct?

Mr. TURNER. Obviously, every building is different, and there is no question, it takes somebody to understand the way the ventilation systems work. If you want to find an area for smokers, it would make sense to have somebody who understands how that ventilation system works to identify the most suitable area.

It will vary from building to building, although there are general principles that would apply and we have got some data sheets that are quite useful for addressing various smoking policies and that

you could apply to any building. You just have to take the general principles that we outline in the data sheet.

Mr. DUNCAN. Dr. LeVois, it has been mentioned several times that cigarette smoking is the major risk factor in lung cancer. And certainly I agree that smoke is a serious detriment to one's health, lung cancer, heart disease, many other things. And I don't want anyone to think that I am encouraging smoking because, not only is there the health risk, but also there is the economic factor.

I have read that many, many poor and lower income people smoke and I think it is sad they spend such a high percentage of their incomes, their limited incomes on tobacco products, you know, when they could be spending those on their children and so forth. But let's get it clear here, you gentleman are not here encouraging smoking, you are just questioning the scientific data on which this EPA report is based; is that correct?

Mr. GORI. That is definitely so, Mr. Duncan.

Mr. DUNCAN. Before you get into that, Dr. Gori, let me just ask you something you could answer very, very quickly. If we say the EPA study has problems, and let's say we disregard anything that—any study you have done. Let's say there is bias there or something. I assume that there have been other major studies on secondhand tobacco smoke and whether or not it is a problem or is a threat.

What do those other—do those other major studies support the EPA reports that recently came out, and if so, how many of those studies have there been, roughly?

Mr. GORI. Well, I am aware of at least one larger study done by the National Academy of Sciences a few years back. However, this study was funded by the EPA, and the EPA report is practically an offshoot of that particular study.

To get back to your original question, I am fully aware of the difficulty that the committee might have in grappling with the different scientific views about the situation here, because again, you are not scientists. But I would hope that you don't consider necessarily that either the EPA or the Surgeon General are infallible.

The issue here is that I am sure you take very seriously your responsibility to legislate on the basis of factual evidence, and therefore I would urge you to look at the facts rather than looking at the messengers.

Mr. DUNCAN. Thank you, Mr. Chairman.

Mr. TRAFICANT. I see in your submitted testimony, Dr. Gori, that the Tobacco Institute has asked you to come here today to explain this. I see in your testimony, Dr. LeVois, that you have been asked by the Tobacco Institute to come here today and testify.

Mr. Turner, were you asked by the Tobacco Institute?

Mr. TURNER. Yes, I was.

Mr. TRAFICANT. Are your expenses being reimbursed by the Tobacco Institute?

Mr. TURNER. Yes.

Mr. LEVOIS. Yes.

Mr. GORI. Yes.

Mr. TRAFICANT. Mr. Turner.

Mr. TURNER. Yes, yes.

Mr. TRAFICANT. Are you being paid in addition to your expenses for testifying here today?

Mr. TURNER. Yes.

Mr. TRAFICANT. Dr. LeVois.

Mr. LEVOIS. Yes.

Mr. TRAFICANT. Dr. Gori.

Mr. GORI. I didn't get the question.

Mr. TRAFICANT. Are you being compensated over and above your actual expenses for testifying today?

Mr. GORI. Oh, yes, yes.

Mr. TRAFICANT. What was that?

Mr. GORI. Yes.

Mr. TRAFICANT. Gentlemen, I appreciate your response, and I appreciate you tolerating some of my questions, and I am sure as we move forward we will be calling not only Mr. Turner back, but perhaps you, as well. We do want to get all the facts together. We appreciate your testimony, and I would say that we would be looking at the indoor air quality as well, Mr. Turner, before this is all over.

Thank you, gentlemen.

With that, ladies and gentlemen, that concludes our hearing.

[Whereupon, at 3:15 p.m., the subcommittee was adjourned.]

[Subsequent to the hearing, additional follow-up questions were submitted to Dr. Gori, and Dr. LeVois by Representative Bill Emerson. The questions and responses follow:]

U.S. HOUSE OF REPRESENTATIVES
COMMITTEE ON PUBLIC WORKS
SUBCOMMITTEE ON PUBLIC BUILDINGS AND GROUNDS

Responses to Questions by Representative Emerson
on H.R. 881

Gio Batta Gori, Ph.D

April 20, 1993

Question 1: "Dr. Gori, in your critique of the EPA report you point out that this is not an isolated instance where the agency has mishandled a scientific issue. If Congress were to bless the agency's behavior by passing this legislation, what kind of message do you think that would send to EPA and to other agencies that are charged with basing policy decisions on sound science?"

I would base my response to this question on the very significant report, "Safeguarding the Future: Credible Science, Credible Decisions," to which I referred briefly in my testimony at the March 11 hearing. This report was issued in 1992 by a panel of distinguished scientists commissioned by former EPA Administrator Reilly to evaluate the agency's problems in dealing with scientific issues.

Perhaps the most striking aspect of the report is that it had to be commissioned at all. EPA is hardly typical in requiring outside help to sort out what may fairly be called a crisis of public confidence. Recall what the Expert Panel had to say: "Currently, EPA science is of uneven quality, and the Agency's policies and regulations are

frequently perceived as lacking a strong scientific foundation." And furthermore, "EPA science is perceived by many people, both inside and outside the Agency, to be adjusted to fit policy." In my testimony, I pointed out how the agency's ETS risk assessment is a textbook illustration of these problems.

From dioxin to Alar, EPA has time and again placed itself in the position of having to backpedal from alarming statements about environmental health effects once the lack of scientific foundation for such statements became clear. No other agency whose mission is to take regulatory action on the basis of sound science has such a dismal record. It is my view that allowing the ETS risk assessment to stand and, indeed, to form the basis for public policy decisions, would send precisely the wrong message to the agency. Rather than forcing itself to confront the sober lessons of the "Safeguarding the Future" report, EPA would instead pay lip service to the report while continuing to lurch from one exaggerated environmental crisis to the next, wasting scarce resources and sowing panic in the bargain.

As a citizen, I am very concerned that allowing EPA to continue without fundamentally altering its behavior will allow the agency to jettison the due process requirement that underlies its mandate. Allowing EPA to regulate and make policy pronouncements without an adequate scientific basis--as it is attempting to do in the case of ETS--would mean that the

agency would be free to choose any pretext to impose its will on the public.

Finally, as a scientist I have a special concern about EPA's behavior. If Congress were to bless EPA's ETS risk assessment by enacting H.R. 881, it would in effect condone careless and shoddy science. As has been the case with so many recent examples, the exaggeration of risk reflected in the EPA report will contribute to the discrediting of science in the eyes of the public, who will no longer know whom or what to believe.

Question 2: "Both Congress and the public are forced to trust the sound judgment of agencies that make decisions on complex scientific issues. What we have seen with regard to the ETS issue is an institutionalized bias that does not repay our trust. What can we do to ensure that this sort of thing does not continue?"

It is an inevitable fact among environmental regulatory agencies that in order to justify their funding, they must be constantly on the alert for new objects of their regulatory zeal. Realistically, I do not think that we will eradicate this tendency. Instead, it must be controlled. This can perhaps best be accomplished through much more

dedicated use of the existing control mechanisms than in the past.

Our objective should be to keep regulatory agencies trustworthy by requiring them to adhere to universally accepted standards of scientific evidence whenever they assert scientific validity as the justification for their regulations. It seems to me that leadership within the agency is the first prerequisite here. The Administration and Congress must ensure that agency appointees will place scientific quality and objectivity above short-term advantage in power or publicity. I hope that Congress will keep this in mind as new EPA appointments are reviewed at the key Assistant Administrator level.

Also, the managers must insist that the agency's scientists provide sound scientific advice--not, as clearly was the case with ETS, whatever justification is desired for a predetermined policy decision. The reward structure for career agency scientists should be geared accordingly.

Other aspects of what might be termed the agency culture should be made to fall in line with the renewed emphasis on scientific objectivity. For example, the review process whereby EPA employs outside scientists to evaluate the quality of agency efforts such as risk assessments obviously is tailor-made to provide an ongoing check on bureaucratic tendencies to slight scientific rigor. That, at least, is the ideal.

Unfortunately, we have witnessed a much less than ideal application of this mechanism in the case of ETS, where the relevant Science Advisory Board panel made no serious effort to review the agency's work comprehensively or objectively. Many of the panelists had either staked out strong antismoking positions that precluded any objective examination of the ETS evidence, or were beholden to EPA for a series of grants and contracts, which obviously works against dispassionate review of agency efforts. The solution to this problem might be to oversee much more carefully the process whereby these panels are constituted, to ensure representation of all credible scientific viewpoints, and to enforce much more rigorous standards regarding conflicts of interest and bias.

SUBCOMMITTEE ON PUBLIC BUILDINGS AND GROUNDS

REGARDING H.R. 881: A BILL TO PROHIBIT SMOKING
IN FEDERAL BUILDINGS

RESPONSE TO WRITTEN QUESTIONS OF CONGRESSMAN BILL EMERSON

BY MAURICE LEVOIS, PH.D.
APRIL 16, 1993

QUESTIONS

1. "Dr. LeVois, you have reviewed the statistical approach that EPA used in its ETS report, and you have heard the criticisms and defenses of that approach that have been offered today. You have had experience in the application of epidemiologic techniques to regulatory problems at another federal agency, the Centers for Disease Control. How does what the EPA did compare with what other agencies do? Is the EPA an aberration, or does the problem of policy-based science occur throughout the public health establishment?"

2. "As a non-scientist, I want to get an understanding of just what it means to shift the standard for statistical significance the way that EPA did. I don't know much about epidemiology, but I do know something about political polls.

(a) It is true, isn't it, that by doubling the possibility of error from 5 percent to 10 percent in the ETS studies the EPA was doing essentially the same thing as doubling the margin of error in a political poll from the typical plus or minus 5 points to plus or minus 10 points?"

(b) I certainly wouldn't want to pay for a political poll that predicted results that could swing 10 points either way. What about health studies with such wide margins of error? Are they worth anything?"

ANSWERS

I hope to explain why I do not believe that the EPA has used statistics appropriately in their ETS risk assessment. My answers to Congressman Emerson's questions can be prefaced by the comments of two recognized authorities in the field of applied biostatistics concerning the use of what scientists refer to as "one-tailed" versus "two-tailed" tests of statistical significance:

J.L. Fleiss - "By performing a one-tailed test, [the researcher] is ruling out as unimportant the possible inference that p_1 [rate of disease in the exposed group] is significantly greater than p_2 [rate of disease in the unexposed group]."

Page 20.

"...a one-tailed test is called for only when the investigator is not interested in a difference in the reverse direction from that hypothesized."

Page 21.

"If, however, the investigator intends to report his results to his professional colleagues, he is ethically bound to perform a two-tailed test."

Page 21.

Joseph L. Fleiss. Statistical Methods for Rates and Proportions. New York: John Wiley & Sons, 1973.

P. Armitage - G. Berry

"The critical value for a one-sided test at level *P* is therefore the same as that for a two-sided test at level 2P. In a sense the distinction is semantic. On the other hand there is often a temptation to use one-sided tests rather than two-sided tests because the probability level is lower, and therefore the apparent significance is greater. A decision to use a one-sided test should *never* be made after looking at the data and observing the direction of the departure. Before the data are examined one should decide to use a one-sided test only if it is quite certain that departures in one particular direction will always be ascribed to chance, and therefore regarded as nonsignificant however large they are. This situation *rarely* arises in practice, and it will be safe to assume that significance tests should almost always be

two-sided." Page 98.

P. Armitage and G. Berry. Statistical Methods in Medical Research. 2nd ed. Oxford, England: Blackwell Scientific Publications Ltd, 1987.

The EPA has not used an appropriate significance test for their meta-analysis of data from selected epidemiologic studies on the relationship of spousal smoking and lung cancer (the central analysis used by EPA to estimate an ETS/lung cancer risk), nor has the EPA adequately addressed fundamental problems in the data used for that analysis.

The first draft of the EPA's ETS risk assessment used a standard two-sided significance test for the main spousal smoking analysis. Had the same two-sided significance test been used in the revised EPA risk assessment the result would not have been statistically significant. In their final risk assessment the EPA shifted to a one-sided statistical test. This shift suggests that the EPA decided to use a one-sided significance test after reviewing the data and observing that a two-sided test did not achieve statistical significance. This is exactly the error that Armitage and Berry warn against.

A test of statistical significance is only an aid to decision-making. Such tests help to describe the level of confidence one has that a given result is not due to chance alone. The 95% or 99% confidence levels for significance tests are standards used by researchers both in federal agencies and the scientific community

in general, in both epidemiology and risk assessment. The 90% confidence level used in EPA's ETS risk assessment would not be accepted by that Agency if it were, for example, submitted by a manufacturer as part of an application for an EPA-regulated pesticide. A one-in-ten error rate is simply not good enough, and should not have been used in the EPA's ETS risk assessment.

Prior to release of the EPA document new spousal smoking data were published in two studies, including the largest U.S. study, that brought the pooled lung cancer risk estimate down even further, to a level that would not be significant at even the 90% confidence level. Such statistical instability plainly contradicts EPA's claim of "medium to high" confidence in their ETS risk estimate, and underscores the need for higher scientific standards than they have employed.

Tests of statistical significance are valid aids in decision-making only if the underlying data are unbiased. The error EPA made by employing a one-sided, 90% significance test should not obscure a more basic and important error. The EPA has pooled results from spousal smoking studies that are not adequate to answer questions about possible health effects of ETS exposure. This is because spousal smoking is a biased proxy for ETS exposure. Spouses of smokers tend to have greater exposure to other lung cancer risk factors than do spouses of nonsmokers. The EPA attributes all of the reported spousal smoking-associated lung cancer risk to ETS exposure, even though it is clear that spouses of smokers tend to have poorer diets, lower socioeconomic status,

and increased occupational exposures, and are more likely to have a history of prior pulmonary illness than are the spouses of nonsmokers. The EPA's ETS meta-analysis simply pools biases contained in the individual studies to produce a biased result.

Biases introduced when spousal smoking is employed as a proxy for ETS exposure could easily be responsible for the very weak reported pooled spousal smoking / lung cancer risk estimate. It is noteworthy that meta-analysis of results of studies that have looked at ETS exposure in the workplace (as opposed to using spousal smoking as the ETS exposure definition) shows no increase in ETS related lung cancer risk. This important discrepancy undermines both the validity of the spousal smoking proxy and the inference that ETS exposure causes lung cancer.

PREPARED STATEMENTS OF WITNESSES

TESTIMONY OF

THE HONORABLE RICHARD J. DURBIN

BEFORE THE

SUBCOMMITTEE ON PUBLIC BUILDINGS AND GROUNDS
COMMITTEE ON PUBLIC WORKS AND TRANSPORTATION

MARCH 11, 1993

Mr. Chairman, I would like to commend you for your interest in the issue of secondhand smoke and thank you for the opportunity to testify today.

It's time for the federal government to extend full protection from secondhand smoke to the workers and visitors in all federal offices, including all office buildings owned or leased by the Executive, Legislative, and Judicial Branches of the U.S. government.

On January 7, after several years of intensive study, the Environmental Protection Agency formally classified environmental tobacco smoke as a Group A carcinogen. This classification is reserved for substances which are known to cause cancer in humans, including asbestos, benzene, and arsenic.

EPA found that secondhand smoke causes approximately 3,000 lung cancer deaths annually in U.S. nonsmokers.

In addition, exposure to secondhand smoke causes 150,000 to 300,000 lower respiratory tract infections such as bronchitis and pneumonia in young children each year, causes additional episodes of asthma and increased severity of asthma symptoms in children who already have asthma, and may be a risk factor for 8,000 to 26,000 new cases of asthma annually in children who would not otherwise become asthmatic.

In response to these facts, I introduced legislation, H.R. 710, to protect federal employees from secondhand smoke in the federal workplace and to protect children from secondhand smoke while they are participating in federally-funded children's programs. I am pleased to have more than 40 cosponsors for this measure, including Mr. Hansen and Mr. Mazzoli as lead cosponsors.

Mr. Chairman, with regard to federal workers, the bills you and I have introduced both have the same goal, the protection of federal employees who count on us to provide them with a safe environment as they serve the American people.

PRINTED ON RECYCLED PAPER

My legislation would prohibit smoking in all federal buildings, except in areas that are ventilated separately from the rest of the building.

The EPA and others who have examined this issue have told us there are only two ways to protect nonsmokers from the hazards of breathing secondhand smoke. Either indoor smoking must be banned, or it must be limited to separately-ventilated smoking areas.

Separate smoking sections that are not separately ventilated are not acceptable, because the smoke recirculates through the building's ventilation system directly into the rooms used by nonsmokers.

My legislation does not require that agencies establish separately-ventilated smoking rooms, nor does it authorize funding to making the engineering changes that might be necessary. However, it leaves open the possibility of separate ventilation in cases where separate ventilation could be accomplished without significant cost.

If we take action on smoking in federal office buildings, we will be following the lead of many private companies throughout the country. In a 1991 survey of members of the Society for Human Resource Management, 34% of the firms that responded indicated they have declared their facilities "smokefree". Five years earlier, only 2% of the firms were smokefree. Today the percentage is undoubtedly even larger.

It is time for the federal government to provide its workers with the same basic protection against secondhand smoke that is offered by a growing number of private sector employers.

Mr. Chairman, I appreciate your desire to act on this issue, and I hope that our colleagues will join us in bringing the smoking policies under federal jurisdiction into conformity with the scientific and health findings of the EPA and the growing practice in private office buildings throughout the country.

Thank you again for bringing attention to this issue.

TESTIMONY OF
DR. WILLIAM FARLAND
DIRECTOR
OFFICE OF HEALTH & ENVIRONMENTAL ASSESSMENT
OFFICE OF RESEARCH AND DEVELOPMENT
U.S. ENVIRONMENTAL PROTECTION AGENCY
BEFORE THE
SUBCOMMITTEE ON PUBLIC BUILDINGS AND GROUNDS OF THE
COMMITTEE ON PUBLIC WORKS AND TRANSPORTATION
U.S. HOUSE OF REPRESENTATIVES

MARCH 11, 1993

Good morning, Mr. Chairman and Members of the Subcommittee. Thank you for the opportunity to appear before you today to discuss H.R. 881, a bill to prohibit smoking in federal buildings.

The U.S. Environmental Protection Agency (EPA) has published a major assessment of the respiratory health risks of passive smoking (*Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders; EPA/600/6-90/006F*). The report concludes that exposure to environmental tobacco smoke (ETS) -- commonly known as secondhand smoke -- is responsible for approximately 3,000 lung cancer deaths each year in nonsmoking adults in the U.S. and seriously affects the respiratory health of hundreds of thousands of children. The following testimony summarizes the development of the report, the scientific review process, and the major findings. It concludes with EPA's endorsement of the objectives of the proposed legislation and the suggestion that the ban be extended to include all federal government vehicles.

Background

In recent years, comparative risk studies performed by EPA and its Science Advisory Board have consistently ranked indoor air pollution among the top five

environmental risks to public health. Environmental tobacco smoke is one of the major indoor air pollutants and, given the known health impact of tobacco smoking, there has been concern that nonsmokers may also be at risk of serious health effects.

As part of its efforts to address all types of indoor air pollution, in 1988, EPA's Indoor Air Division requested that EPA's Office of Research and Development (ORD) undertake an assessment of the respiratory health effects of passive smoking. The report was prepared by ORD's Office of Health and Environmental Assessment.

The document has been prepared under the authority of Title IV of Superfund (The Radon Gas and Indoor Air Quality Research Act of 1986), which directs EPA to conduct research and disseminate information on all aspects of indoor air quality.

Public and Scientific Reviews

A draft of this assessment was released for public review and comment in June 1990. In December 1990, EPA's Science Advisory Board (SAB), a committee of independent outside scientists, conducted a review of the draft report and submitted its comments to the EPA Administrator in April 1991. In its comments, the SAB's Indoor Air Quality/Total Human Exposure Committee concurred with the primary findings of the report, but made a number of recommendations for strengthening it.

Incorporating recommendations from both the public and the SAB, a revised draft was transmitted to the SAB in May 1992 for a second review. Following a July 1992 meeting the SAB panel endorsed the report and its conclusions, including a unanimous endorsement of the classification of environmental tobacco smoke as a Group A (known human) carcinogen.

The EPA also received and reviewed public comments on the second draft, and integrated all appropriate material into the final risk assessment. The final report was released in January 1993, at a joint press conference held by former Administrator Reilly and former Department of Health and Human Services Secretary Sullivan.

Major Conclusions

Based on the weight of the available scientific evidence, EPA has concluded that the widespread exposure to environmental tobacco smoke in the U.S. presents a serious and substantial public health risk.

In adults:

* ETS is a human lung carcinogen, responsible for approximately 3,000 lung cancer deaths annually in U.S. nonsmokers. ETS has been classified as a known human or Group A carcinogen under EPA's carcinogen assessment guidelines. This classification is reserved for those compounds or mixtures which have the strongest data to determine a cause - and - effect relationship, including data from human populations. Only ten other agents, including asbestos and radon, have been classified by EPA as Group A carcinogens, and ETS is the only one for which cancer has been observed at typical non-occupational environmental levels.

* ETS has subtle but significant effects on the respiratory health of nonsmokers, including coughing, phlegm production, chest discomfort, and reduced lung function.

In children:

* ETS exposure increases the risk of lower respiratory tract infections such as bronchitis and pneumonia. EPA estimates that between 150,000 and 300,000 of these cases annually in infants and young children up to 18 months of age are

attributable to exposure to ETS. Of these, between 7,500 and 15,000 will result in hospitalization.

* ETS exposure increases the prevalence of fluid in the middle ear, a sign of chronic middle ear disease. Fluid in the middle ear is the major cause of hospitalization of young children for an operation in the U.S.

* ETS exposure in children irritates the upper respiratory tract and is associated with a small but significant reduction in lung function.

* ETS exposure increases the frequency of episodes and severity of symptoms in asthmatic children. The report estimates that 200,000 to 1,000,000 asthmatic children have their condition worsened by exposure to environmental tobacco smoke.

* ETS exposure is a risk factor for new cases of asthma in children who have not previously displayed symptoms.

Scientific Approach

EPA reached its conclusions concerning the potential for ETS to act as a human carcinogen based on an analysis of all of the available data, including more than 30 epidemiologic (human) studies looking specifically at passive smoking and lung cancer in nonsmokers. In addition, EPA considered information on active smoking, animal data, biological measurements of human uptake of environmental tobacco smoke components, and other available data. The conclusions on lung cancer were based on what is commonly known as the total "weight-of-evidence," rather than on any one study or type of study.

Specifically, the finding that ETS should be classified as a Group A carcinogen is based on the conclusive evidence of the dose-related lung carcinogenicity of mainstream smoke in active smokers, the chemical similarities of mainstream smoke

and the sidestream smoke given off by the burning end of the cigarette and the known exposure and uptake of ETS at levels which could increase risk. The finding is bolstered by the statistically significant exposure-related increase in lung cancer in nonsmoking spouses of smokers which is observed in an analysis of more than 30 epidemiology studies, from eight different countries, that examined the association between secondhand smoke and lung cancer.

The weight-of-evidence analysis for the noncancer respiratory effects in children is based primarily on a review of more than 100 studies, including over 50 recent epidemiology studies of children whose parents smoke.

Other Assessments of ETS and Respiratory Disease

The EPA's conclusions on the respiratory effects of passive smoking strengthen and confirm those of earlier assessments by the U.S. Surgeon General (1986), and the National Research Council of the National Academy of Sciences (1986). The World Health Organization has also concluded that ETS causes excess risk from lung cancer (1986) and other respiratory disorders (1992). The National Institute of Occupational Safety and Health (1991) concluded that occupational exposure to ETS causes increased risks of lung cancer and probably heart disease. The position of the National Cancer Institute (1993) is that ETS is a proven cause of lung cancer in nonsmoking adults and is associated with an increased risk of coronary heart disease.

To my knowledge only the tobacco industry and those associated with it continue to deny the serious health risk posed by exposure to ETS.

Policy Implications

What are the implications of this report for public policy? I believe we should first put the risks associated with ETS into perspective. The EPA estimates that about 20 to 30 percent of all lung cancers caused by factors other than a cancer victim's own smoking are attributable to environmental tobacco smoke. The increased risk of dying from lung cancer is about 1-in-1,000 from all ETS exposures outside the home. Exposure to ETS varies, but higher exposures are associated with higher risks. For example, people whose spouses smoke in the home face an average increased risk of 2-in-1,000.

Risks in this range are considered high. For comparison, EPA generally sets its standards or regulations so that increased risks are below 1-in-10,000 to 1-in-1 million. The additional risks on childhood respiratory health make an even more compelling case for appropriate actions to control involuntary exposures.

With this report, together with the 1991 NIOSH report, we believe that we have established a firm scientific foundation for such actions. While EPA has no regulatory authority over ETS, we intend to work with other Federal agencies to ensure that the issue is adequately addressed. For instance, the OSHA, which has statutory authority for worker safety and health, published a Request for Information (RFI) in September 1991 on Indoor Air Quality Issues, such as ETS. The Agency is currently reviewing the more than 1240 responses to the RFI, including EPA's risk assessment report, in order to determine further Agency action regarding smoking in the workplace. Also, in cooperation with other government agencies, EPA will carry out an education and outreach program over the next two years to inform the public and policy makers on what to do to reduce the health risks of ETS as well as other indoor air pollutants.

I would also personally recommend that the provisions of the bill be extended to include a ban on smoking in all vehicles owned or leased by the Federal Government. Studies have shown that smoking in smaller confined spaces can dramatically increase ETS concentrations over those normally seen in homes or buildings.

I believe that the general thrust of H.R. 881 represents responsible governmental action for protecting Public Health and will provide a model for State and local governments to follow. EPA therefore supports the objectives of H.R. 881, while defering to interested agencies regarding details of the bill.

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Lung Cancer in Nonsmoking Women: A Multicenter Case-Control Study[1]

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Abstract

The association between exposure to environmental tobacco smoke and lung cancer in female lifetime nonsmokers was evaluated using data collected during the first 3 years of an ongoing case-control study. This large, multicenter, population-based study was designed to minimize some of the methodological problems which have been of concern in previous studies of environmental tobacco smoke and lung cancer. Both a cancer control group and a population control group were selected in order to evaluate recall bias. A uniform histopathological review of diagnostic material was conducted for case confirmation and detailed classification. Biochemical determination of current exposure to tobacco and screening of multiple sources of information to determine lifetime nonuse were utilized to minimize misclassification of smokers as nonsmokers.

A 30% increased risk of lung cancer was associated with exposure to environmental tobacco smoke from a spouse, and a 50% increase was observed for adenocarcinoma of the lung. A statistically significant positive trend in risk was observed as pack-years of exposure from a spouse increased, reaching a relative risk of 1.7 for pulmonary adenocarcinoma with exposures of 80 or more pack-years. The predominant cell type of the reviewed, eligible lung cancer cases was adenocarcinoma (78%). Results were very similar when cases were compared to each control group and when separate analyses were conducted for surrogate and personal respondents. Other adult-life exposures in household, occupational, and social settings were each associated with a 40–60% increased risk of adenocarcinoma of the lung. No association was found between risk of any type of lung cancer and childhood exposures from a father, mother, or other household members.

Introduction

Approximately one decade has passed since the initial reports of increased risk of lung cancer in nonsmoking women married to smokers (1, 2). The ensuing studies have provided a body of data which suggests a small but significant elevation in risk of lung cancer associated with exposure to ETS[3] (3–22). In reported prospective studies exposure has been assessed by the spouse's smoking history, primarily that of husbands. In case-control studies, the primary ETS exposure assessed has also been that from a spouse, although exposures from parents, other household exposures, and the workplace have been examined in some studies.

In general, these studies have included fewer than 100 nonsmoking lung cancer cases whose self-reported smoking status has not been validated by biochemical determination or other means. Reviews of available studies of ETS and lung cancer in nonsmokers by the National Research Council (23), the International Agency for Cancer Research (24), and others (25, 26) have concluded that although misclassification is unlikely to account for all of the observed increased risk, some misclassification of current or former smokers as nonsmokers is likely (0.5–5.0%). Because smokers tend to marry smokers, misreporting may introduce some bias in the estimation of the magnitude of the observed effect.

This study was undertaken in 1985 in an effort to address a number of unresolved issues related to ETS:

(a) Misclassification of Smoking Status. Multiple sources of information are utilized to ascertain nonsmoking status (medical record, physician, and then the study subject or surrogate). Study respondents are questioned twice (at contact to set up the interview and at the beginning of the interview). Self-reported current nonsmoking status is corroborated by measurement of urinary cotinine.

(b) Histopathological Specificity. Microscopic diagnostic slides are reviewed by one pulmonary pathologist both to confirm eligibility of cases as primary lung carcinomas and to provide a detailed review (subtype, differ-

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[1] This research was supported by Grant CA40095 from the National Cancer Institute.

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[3] The abbreviations used are: ETS, environmental tobacco smoke; SEER, Surveillance, Epidemiology, and End Result; OR, odds ratio; CI, confidence interval.

entiation) and classification of the histopathological cell type.

(c) Recall Bias. Two control groups, one with colon cancer and one from the general population, are selected for case-control comparisons. Differential recall between cases and colon cancer controls should be minimized since both groups are similarly motivated to recall earlier exposures.

(d) Source of ETS Exposure. Information on childhood exposures from a father, mother, and other household members and adult exposures from husband(s), other household members, and occupational and social settings is obtained by questionnaire. The risk associated with exposure to ETS from different sources and during different time periods can be evaluated.

(e) Confounders and Other Risk Factors. Because the magnitude of the main ETS effect is expected to be small, it is important to take into account potential confounding factors and effect modifying factors in a study with a sufficiently large number of cases and controls. It is anticipated that upon completion of this study about 600 cases and twice that number of controls will have participated.

This report represents findings from the ongoing study and includes the largest number of lifetime nonsmokers with lung cancers reported to date. This report was felt to be justified given the public health importance of the issue under investigation.

Methods

The study is a population-based case-control study of lung cancer in women who have never used any tobacco product. This preliminary report includes cases diagnosed during the first three years (December 1, 1985 through December 31, 1988) of a 5-year study. At the time of diagnosis cases were residents of one of five major metropolitan areas throughout the United States, including Atlanta (Clayton, Cobb, DeKalb, Fulton, and Gwinnett counties), Houston (Galveston and Harris counties), Los Angeles (Los Angeles County), New Orleans (Jefferson, Orleans, and St. Bernard parishes), and the San Francisco Bay Area (Alameda, Contra Costa, Marin, San Francisco, San Mateo, and Santa Clara counties), representing a population of approximately 18.5 million people or 8% of the U. S. population.

Case and Control Selection

Rapid case ascertainment procedures, which included review of pathology reports from study hospitals, were utilized to identify potentially eligible lung cancer cases. Eligible cases included English-, Spanish-, or Chinese-speaking females, aged 20–79, who had a histopathologically confirmed diagnosis of primary carcinoma of the lung (International Classification of Disease, 9th Revision, code 162) made prior to death, had no history of previous cancer, and who were lifetime nonusers of tobacco. Lifetime nonusers of tobacco are defined for this study as persons who had smoked fewer than 100 cigarettes and had not used any other form of tobacco for more than 6 months.

Two control groups were selected. The first control group, referred to as the population control group, was selected by random digit dialing and supplemented by random sampling from the files of the Health Care Financing Administration for women aged 65 and older. Controls were frequency matched to cases on age (<50, 50–59, 60–69, 70+ years) in a 2:1 control:case ratio. They met the same eligibility criteria as cases for age, residence, language, and tobacco use.

Females, aged 20–79, with a diagnosis of primary carcinoma of the colon (International Classification of Disease, 9th Revision, code 153) who met the language, previous cancer, lifetime nonsmoking, and residential eligibility criteria of the cases, were identified and frequency matched to lung cancer cases by 10-year age groups and race. This second control group was selected because there is no established increased risk of colon cancer associated with either active or passive smoking, and it provided an opportunity to examine the issue of recall bias associated with a recent diagnosis of cancer.

A multistep procedure was used to determine lifetime smoking status. After identification of a potentially eligible lung cancer case or colon cancer control, the hospital chart was reviewed to obtain demographic data and available information on tobacco use. Patients identified as current or former smokers in the medical record were considered ineligible. In study areas where individual physician notification was required/preferred, the tobacco use history was requested from the physician for potentially eligible cases and colon controls identified as nonusers of tobacco or with unknown smoking status according to the hospital record. Women who were identified as current or former smokers by their physicians were considered ineligible. All remaining cases and colon cancer controls believed to be nonsmokers or with unknown smoking status were contacted by telephone to elicit information on tobacco use. Women who reported ever smoking 100 or more cigarettes or using any other form of tobacco for more than 6 months were considered ineligible. The identical telephone screening procedure was used for the population control group. At the time of the interview, the tobacco use screening questions were repeated to confirm each study subject's reported nonuse of tobacco.

The questionnaire was translated from English into Spanish and Chinese, and interviewers fluent in those languages conducted the non-English as well as English interviews. Interviews were completed for 431 of 514 incident cases (84%), 358 of 489 colon cancer controls (73%), and 794 of 1105 population controls (72%). Sixty-one (3.8%) of the interviews were conducted in Spanish (n = 14) or Chinese (n = 47): 22 cases (5%); 23 colon cancer controls (6%); and 16 population controls (1.5%). A next-of-kin interview was solicited for lung cancer cases and colon cancer controls who were too ill or deceased. All population controls were self-respondents because of the sampling method used to identify these controls. A total of 143 lung cancer case interviews and 35 of 352 colon cancer control interviews were conducted with next-of-kin respondents, representing 34% and 10% of the eligible respondents.

An extensive structured questionnaire obtained information concerning household, occupational, and other exposures to environmental tobacco smoke during the study subject's lifetime. Data were also collected on lifetime occupational history, usual adult diet, family and personal medical histories, and other exposures of interest, which are not included in this report.

Table 1 Urinary cotinine/creatinine (ng/mg) by case-control status

	Lung cancer cases	Colon cancer controls	Population controls
Status			
Completed	239	260	684
Eligible (<100 ng/mg)	237	253	670
Ineligible (≥100 ng/mg)	2	7	14
Not performed			
Self-respondents	58	66	110
Next-of-kin respondents	134	32	
Results			
Eligible (<100 ng/mg)			
Mean (SD)	6.95 (12.11)	5.82 (11.68)	9.68 (12.88)
Median	2.0	0	5.4
Range	0-71.4	0-88.4	0-95.01
Ineligible (≥100 ng/mg)			
Range	131-219	145-5,163	103-14,014

Table 2 Pathology review

Reviewed, found to be eligible	359 (85%)
Adenocarcinoma	281
Large cell carcinoma	43
Squamous cell carcinoma	20
Small cell carcinoma	12
Others and not otherwise specified	3
Not reviewed/insufficient material	50 (12%)
Histology by hospital pathologist	
Adenocarcinoma	30
Large cell carcinoma	5
Squamous cell carcinoma	7
Small cell carcinoma	2
Others and not otherwise specified	6
Review pending	11 (3%)
Total cases	420
Reviewed, found to be ineligible	9

Eligibility Review Procedures

Biochemical Determination of Current Tobacco Use. Cotinine, a major metabolite of nicotine, is an indicator of recent exposure to tobacco (27). Urinary cotinine was used to corroborate self-reported current nonsmoking status of study subjects. A urine sample was collected from all consenting study subjects at the time of interview. The specimens were stored at −20°C until shipment to the American Health Foundation for analysis.

Cotinine was quantitated by radioimmunoassay using the method of Haley *et al.* (28) with a modification of the antibody of Langone *et al.* (29). Cotinine levels were adjusted for urine flow based on creatinine values by determining the cotinine/creatinine ratio. Creatinine was determined by spectrophotometry using the Kodak Ektachem 400 Clinical Chemistry Analyzer.

At this time biochemical analysis is complete for 239 of 431 cases (55.5%), 260 of 358 colon cancer controls (72.6%), and 684 of 794 population controls (86.1%) (Table 1). Two of 239 case samples (0.8%), 7 of 260 colon cancer control samples (2.6%), and 14 of 684 population control samples (2.0%) had cotinine/creatinine levels of 100 ng/mg or greater. There is no established cotinine/creatinine level which clearly discriminates smokers from true nonsmokers heavily exposed to ETS. Under relatively high levels of exposure to ETS in aircraft and in exposure chambers, urinary excretion has reached a level of 55 ng/mg creatinine (30, 31). In this study, women whose cotinine/creatinine level exceeded 100 ng/mg were excluded from the study to eliminate persons likely to be active smokers, while allowing for the possibility of very high ETS exposures reflected in urinary levels of 56–99 ng/mg creatinine. Had the lower value of 55 ng/mg been selected as a cutpoint to avoid possible misclassification of active smokers as nonsmokers, 4 additional cases (1.6%), 2 colon cancer controls (0.8%), and 13 population controls (1.9%) would have been excluded from the analyses, with negligible effect on the results.

Histopathological Review. Representative diagnostic microscopic tissue slides for each case were requested from the hospital. These slides were reviewed by one pathologist specializing in pulmonary pathology. A total of 368 of 429 (86%) potential cases have undergone review. As shown in Table 2, 359 (98%) of the reviewed cases were confirmed as primary bronchogenic carcinoma. The histopathological primary cell type of the eligible cases is as follows: adenocarcinoma, 78%; large cell carcinoma, 12%; squamous cell carcinoma, 6%; small cell carcinoma, 3%; others, 1%. The histopathological cell type distributions were similar in the five study centers.

The overall concordance between the review pathology diagnosis and the original hospital pathology diagnosis was 81% (Table 3). The concordance varied greatly by histopathological cell type. Ninety-seven % (237 of 244) of the cases originally classified as adenocarcinomas were confirmed as this histopathological type upon review. Similarly, 10 of 11 (91%) of small cell carcinomas were so classified upon review. Concordance rates of 56% and 67% were seen for large cell and squamous cell carcinomas, respectively. A relatively large proportion of cases originally classified as large cell or squamous cell carcinomas were classified as adenocarcinomas by the review pathologist: 18 of 46 (39%) and 6 of 24 (25%), respectively. Based on hospital pathology reports, 34 subjects were categorized as "other primary lung carcinomas" which primarily included diagnoses of poorly differentiated carcinoma, bronchogenic carcinoma not otherwise specified, or malignant cells not otherwise specified. Upon review, 94% of these cases were classified into more specific histopathological cell types.

The nine cases (2%) found not to have primary bronchogenic carcinoma on review were excluded from all analyses. Three of these nine cases were determined to be carcinoid tumors, two were lymphomas, three were carcinomas metastatic to the lungs from other primary sites, and one was a benign neoplasm. The 61 cases that have not undergone histopathological review are included in analyses of all lung cancers combined (n = 420) but are not included in analyses stratified by histopathological type.

Statistical Analyses

Exposure to ETS was examined by source. Sources include both adult and childhood exposures as follows: spouse, other household members; occupational ETS exposures; and social or leisure time (nonhousehold, nonoccupational) ETS during adult life; and father, mother, and other household members who lived in the

Table 3 Distribution of lung cancer histopathological cell types by hospital diagnosis and review diagnosis

Review diagnosis	Hospital diagnosis					
	Adenocarcinoma	Large cell carcinoma	Squamous cell carcinoma	Small cell carcinoma	Other lung carcinoma	Total
Adenocarcinoma	237	18	6	1	19	281
Large cell carcinoma	6	26	1	0	10	43
Squamous cell carcinoma	0	2	16	0	2	20
Small cell carcinoma	0	0	1	10	1	12
Other primary lung carcinomas	1	0	0	0	2	3
Total	244	46	24	11	34	359

home 6 months or more during childhood. Childhood was defined as the first 18 years of life. Exposures from parents after that time were classified as other household members during adult life. Dichotomous ETS exposures were first examined (ever or never) by type of tobacco: cigarettes; pipe; cigar; or any of these types of tobacco. Dose was estimated, as appropriate, by intensity (e.g., number of cigarettes/day), duration (e.g., number of years exposed), or a combination (e.g., pack-years). Pack-years of cigarette exposure from the spouse were calculated by multiplying the number of packs smoked per day by the number of years the spouse smoked cigarettes while living with the study subject. Pack-years of exposure were summed for all smoking spouses of each study subject.

One of the objectives of this study was to evaluate the association of ETS with specific histopathological cell types of lung cancer. The skewed distribution of histopathological types precluded any meaningful analysis by specific cell type other than adenocarcinoma and all other cell types combined. The results are presented for all lung cancers combined ($n = 420$) and adenocarcinomas confirmed by histopathological review ($n = 281$).

Cases were compared to each control group with regard to the distribution of relevant covariates such as age, education, income, and race/ethnicity. The association of ETS exposure with lung cancer risk was investigated first in contingency tables stratified by design or sampling variables (age, race, study center) and relevant covariates. Summary adjusted odds ratios and test statistics were calculated by the method of Mantel and Haenszel (32). Unconditional logistic regression analyses were then used to estimate the associations by summary adjusted odds ratios, confidence limits, and test statistics (33, 34).

Results

Demographic characteristics of cases and controls are presented in Table 4. Cases and controls were similar with respect to matching variables and most demographic variables. The largest number of cases ($n = 160$, 38%) were residents of Los Angeles, followed by cases from the San Francisco Bay Area ($n = 149$, 35%), and then the three smaller study centers in the southern United States: Atlanta ($n = 46$, 11%); Houston ($n = 39$, 9%); and New Orleans ($n = 26$, 6%).

The age distribution of cases and controls is uniform, with 73 to 74% of each series between the ages of 60 through 79. The proportion of older women in this group of female nonsmokers with lung cancer is higher than that among all female lung cancer cases in the SEER Program 1974–1986, in which only 48% of the cases were aged 65 or older (35).

Cases tended to have a somewhat lower household income and less education than the population controls. Approximately 35% of cases and controls spent their childhood in cities with populations of 50,000 or more, and the majority of cases and controls (70%, 68%, 77% for cases, colon cancer controls, and population controls, respectively) resided in cities during most of their adult life.

The estimated risks of lung cancer in nonsmoking women associated with ever having lived with a spouse who smoked are presented in Table 5. The adjusted ORs and the 95% CI are very similar for all spouse-related exposures regardless of control group. For all histopathological types of lung cancer combined, a 30% increase in risk is observed (OR = 1.28 and 1.29 with colon cancer and population controls). For each of the three types of tobacco smoked, the ORs ranged from 1.14 to 1.26. When the case series is restricted to the 281 pulmonary adenocarcinomas confirmed by histopathological review, the association is more pronounced. Approximately 50% elevations in risk of adenocarcinomas of the lung ($P < 0.05$) are associated with any use of tobacco by spouse(s), and cigarette smoking accounts for most of the tobacco use. The estimated relative risk of pulmonary adenocarcinoma associated with cigarette smoking by spouses was 1.36 (1.02–1.84) with the population controls as comparison and 1.31 (0.94–1.84) with the colon cancer controls as comparison. No association between spouses' tobacco use and lung cancers other than adenocarcinoma (squamous cell, small cell, large cell, and other; $n = 78$) was observed.

Separate analyses were conducted for subjects who personally responded and for whom information was obtained from surrogate respondents. The odds ratios for involuntary exposure to ETS were very similar for both groups of respondents; therefore, the results are not presented in the tables separately by type of respondent. One such example is the estimated relative risk of pulmonary adenocarcinomas associated with cigarette smoking by the spouse: OR = 1.38 and 1.30 for surrogate and self-respondents, respectively, comparing cases to colon cancer controls.

Effects by study center were also examined. The odds ratios by center ranged from a low of 1.17 to a high of 2.64 for risk of pulmonary adenocarcinoma associated with spouses' cigarette smoking. Because of the limited sample sizes, none of the individual study center esti-

Table 4 Distribution of lung cancer cases and controls according to selected demographic characteristics

	Lung cancer cases (n = 240)		Colon cancer controls (n = 351)		Population controls (n = 780)	
	No.	(%)	No.	(%)	No.	(%)
Study center						
Atlanta	46	(11.0)	44	(12.5)	76	(9.7)
Houston	39	(9.3)	35	(10.0)	24	(3.1)
Los Angeles	160	(38.1)	125	(35.6)	358	(45.9)
New Orleans	26	(6.2)	18	(5.1)	44	(5.6)
San Francisco Bay Area	149	(35.5)	129	(36.7)	278	(35.6)
Respondent						
Study subject	277	(66.0)	316	(90.1)	780	(100.0)
Next of kin	143	(34.0)	35	(9.9)		
Age (years)						
20–29	5	(1.2)	1	(0.3)	9	(1.2)
30–39	11	(2.6)	13	(3.7)	42	(5.4)
40–49	23	(5.5)	22	(6.3)	30	(3.9)
50–59	73	(17.3)	55	(15.6)	121	(15.5)
60–69	147	(35.0)	105	(29.8)	221	(28.3)
70–79	161	(38.3)	155	(44.0)	357	(45.8)
Race/ethnic group						
White	266	(63.3)	240	(68.5)	503	(64.5)
Black	44	(10.5)	59	(16.8)	107	(13.7)
Hispanic	32	(7.6)	14	(4.0)	42	(5.4)
Asian	67	(16.0)	35	(10.0)	113	(14.5)
Other	11	(2.6)	2	(0.6)	13	(1.7)
Unknown/refused to answer	0	(0.0)	1	(0.2)	2	(0.4)
Annual income						
<$8,000	72	(17.1)	60	(17.1)	98	(12.6)
$8,000–12,999	63	(15.0)	52	(14.8)	115	(14.7)
$13,000–19,999	48	(11.4)	48	(13.7)	110	(14.1)
$20,000–34,999	73	(17.4)	61	(17.4)	153	(19.6)
$35,000–49,999	37	(8.8)	49	(14.0)	82	(10.5)
≥$50,000	59	(14.1)	35	(10.0)	128	(16.4)
Unknown/refused to answer	68	(16.2)	46	(13.1)	94	(12.0)
Education						
Less than high school	135	(32.1)	84	(23.9)	165	(21.2)
High school	140	(33.3)	134	(38.2)	246	(31.5)
Some college	71	(16.9)	74	(21.1)	181	(23.2)
College	33	(7.9)	28	(8.0)	107	(13.7)
Graduate	25	(6.0)	22	(6.3)	69	(8.9)
Unknown	16	(3.8)	9	(2.6)	12	(1.5)
Usual childhood residence						
Farm	93	(22.1)	78	(22.2)	131	(16.8)
Rural area	49	(11.7)	36	(10.3)	61	(7.8)
<20,000 population	92	(21.9)	81	(23.1)	196	(25.1)
20,000–49,999 population	37	(8.8)	46	(13.1)	98	(12.6)
≥50,000 population	146	(34.8)	109	(31.1)	291	(37.3)
Unknown	3	(0.7)	1	(0.3)	3	(0.4)
Usual adult residence						
Farm	23	(5.5)	15	(4.3)	10	(1.3)
Rural area	10	(2.4)	6	(1.7)	13	(1.7)
<20,000 population	39	(9.3)	28	(8.0)	45	(5.8)
20,000–49,999 population	53	(12.6)	61	(17.4)	108	(13.9)
≥50,000 population	293	(69.8)	240	(68.4)	601	(77.0)
Unknown	2	(0.5)	1	(0.3)	3	(0.4)

mates were statistically significant, and they did not significantly differ from one another.

Estimates of relative risk associated with the number of cigarettes smoked by a spouse were significantly elevated only in the highest exposure category, 40 or more cigarettes/day: 2.06 (1.19–3.54) and 1.69 (1.28–2.61) for adenocarcinoma of the lung comparing cases to colon cancer and population controls, respectively. Odds ratios were similar, although slightly lower, for all types of lung cancer combined: 1.70 (1.02–2.84) and 1.36 (0.90–2.06).

Pack-years were examined as a combined measure of duration and dose of exposure to the husband's cigarette smoking. The odds ratios for all cell types of lung cancer combined and for adenocarcinoma of the lung are displayed in Fig. 1. Separate analyses were conducted with each control group for comparison. Because the findings were so similar for each group, the results are presented for the two control series combined (n = 1131). An increasing risk of lung cancer and adenocarcinoma of the lung associated with an increasing level of exposure to the spouse's cigarette smoking was found. The positive trend in risk by pack-years of exposure is statistically significant for adenocarcinoma of the lung (P < 0.01). A weaker dose response is observed when all histopathological types of lung cancer are combined (trend, P = 0.07).

Exposure to ETS from various sources during adult life was evaluated. The results are summarized in Table 6. For simplicity of presentation, the data in this table also represent the findings using the two control groups combined because the individual results using each control group were entirely consistent. Exposures to cigarette smoking from spouse(s), other household members, on the job and in other activities of adult life ("social") are each associated with an overall 40–60% significant elevation in the risk of adenocarcinoma of the lung. As noted previously for spouse-related exposures, the risk estimates for all lung cancers without regard to cell type tend to be slightly lower than the comparable estimates for adenocarcinoma of the lung. Significant positive trends (P < 0.05) in risk of adenocarcinoma of the lung were associated with increasing duration (years) of exposure to cigarette smoke from a spouse, other household members, and social occasions. For adult household exposures from a spouse and others, estimates of risk rose from lowest to highest in the 30 or more years of exposure category; however, trends were not smooth for exposures in occupational and social settings.

No association was found between risk of any type of lung cancer and childhood exposure to cigars, pipes, cigarettes, or all types of tobacco combined. Table 7 presents the estimated relative risks of lung cancer and adenocarcinoma of the lung among nonsmoking women whose father, mother, or other household member smoked during childhood. None differed significantly from unity. Years of exposure and amount smoked were also examined. No significant elevations in risk were found at any level of smoking by household members during childhood.

Discussion

One of the most striking findings of this study is the distribution of the histopathological cell types of lung cancer in a population-based series of cases well screened to determine lifetime nonsmoker status. Seventy-eight % of 359 reviewed eligible cases in this report were classified as adenocarcinomas. This high proportion of adenocarcinomas and the paucity of squamous and small cell carcinomas was consistent across all study

Table 5 Association between smoking status of spouse(s) and lung cancer risk[a]: all lung cancer and adenocarcinoma of the lung

Spouse ever smoked tobacco (by type)	Cases	Colon cancer controls	Population controls	Adjusted odds ratio[a]	
				Colon cancer controls OR (95% CI)	Population controls OR (95% CI)
All lung carcinomas	(*n* = 420)	(*n* = 351)	(*n* = 780)		
Any type of tobacco	294	231	492	1.28 (0.93–1.75)	1.29 (0.99–1.69)
Cigarettes	264	209	441	1.17 (0.87–1.59)	1.20 (0.93–1.55)
Cigars	64	54	97	1.14 (0.76–1.71)	1.26 (0.88–1.80)
Pipe	63	52	110	1.17 (0.78–1.77)	1.21 (0.85–1.72)
Adenocarcinoma	(*n* = 281)	(*n* = 351)	(*n* = 780)		
Any type of tobacco	203	231	492	1.44 (1.01–2.05)[b]	1.47 (1.08–2.01)[b]
Cigarettes	184	209	441	1.31 (0.94–1.84)	1.36 (1.02–1.84)[b]
Cigars	41	54	97	1.05 (0.67–1.66)	1.15 (0.76–1.74)
Pipes	44	52	110	1.16 (0.74–1.82)	1.20 (0.81–1.79)

[a] Adjusted for age (continuous), race (white, black, other), study area (Los Angeles, San Francisco Bay Area, Southern U.S.: Atlanta, Houston, and New Orleans), annual family income (<$13,000, $13,000–$34,999, $35,000+), and education (<high school degree, high school degree, some college or higher).
[b] $P < 0.05$.

centers. In the study of Kabat and Wynder (8), a similar proportion (74%) of Kreyberg II type tumors was found in their series of 97 nonsmoking females whose self-reported nonsmoking status was confirmed by chart review. In the United States adenocarcinoma is the most common histopathological cell type of primary lung cancer in women, but the proportion of all female lung cancer cases with all subtypes of adenocarcinomas (papillary, acinar, bronchioloalveolar, and solid) is 34% (SEER Public User Tape, 1978–1987).

Our study, in which adenocarcinoma is predominant and is the cell type clearly associated with increased risk from adult ETS exposures, is in contrast to several of the earlier studies of involuntary exposure to ETS. Trichopoulos *et al.* (2) in the initial case-control study of lung cancer and passive smoking among nonsmoking women excluded cases of adenocarcinoma including bronchioloalveolar; however, that study included no histopathological review. They reported an odds ratio from 1.8 to 3.4 associated with the husband's smoking habits. Dalager *et al.* (16) reported a 3-fold elevated risk associated with the spouse's smoking only for squamous and small cell carcinomas and no increased risk of other cell types, of which adenocarcinoma and its subtype, bronchioloal-

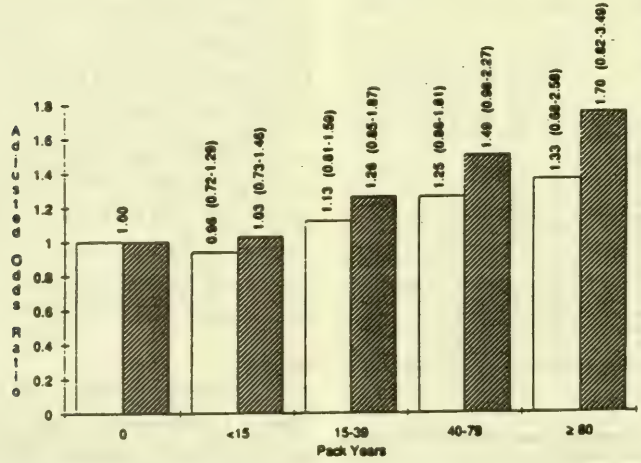

Fig. 1 Adjusted odds ratios for all lung cancer and for adenocarcinoma of the lung associated with pack-years of exposures from spouse(s). □, all lung cancer, trend $P = 0.07$; ■, adenocarcinoma, trend $P < 0.01$.

Table 6 Association between risk[a] of lung cancer and adult exposures to cigarette smoke among nonsmoking women

Years of exposure by source	All lung carcinomas adjusted odds ratio[a] (95% CI)	Adenocarcinoma of the lung adjusted odds ratio[a] (95% CI)
Household exposure		
Spouse		
Ever exposed[b]	1.21 (0.96–1.54)	1.38 (1.04–1.83)[c]
0 years	1.00	1.0
1–15	1.19 (0.88–1.61)	1.33 (0.93–1.89)
16–30	1.14 (0.82–1.59)	1.40 (0.96–2.05)
>30	1.25 (0.91–1.72)	1.43 (0.99–2.09)
	Trend $P = 0.14$	Trend $P = 0.03$
Other household members		
Ever exposed[b]	1.23 (0.97–1.56)	1.39 (1.05–1.82)[c]
0 years	1.00	1.00
1–5	1.20 (0.90–1.61)	1.36 (0.98–1.89)
6+[d]	1.23 (0.89–1.69)	1.35 (0.93–1.94)
	Trend $P = 0.12$	Trend $P = 0.04$
Occupational exposure		
Ever exposed[b]	1.34 (1.03–1.73)[c]	1.44 (1.06–1.97)[c]
0 years	1.00	1.00
1–15	1.23 (0.86–1.77)	1.58 (1.05–2.39)[c]
16–30	1.45 (1.05–2.00)[c]	1.42 (0.97–2.07)
>30	1.30 (0.93–1.80)	1.37 (0.92–2.02)
	Trend $P = 0.02$	Trend $P = 0.10$
Social exposure[e]		
Ever exposed[b]	1.58 (1.22–2.04)[f]	1.60 (1.19–2.14)[f]
0	1.00	1.00
1–15	1.34 (0.97–1.84)	1.29 (0.89–1.87)
16–30	2.01 (1.29–3.15)[f]	2.40 (1.47–3.90)[f]
>30	1.65 (0.98–2.80)	1.50 (0.78–2.77)
	Trend $P = 0.0006$	Trend $P = 0.002$

[a] Adjusted for age, race, study area, annual income, and education.
[b] Referent: never exposed.
[c] $P < 0.05$.
[d] Too few subjects exposed 16+ years.
[e] Social exposure is defined as exposure of 2 or more h/week from sources other than occupational and household members, including spouse.
[f] $P < 0.01$.

veolar carcinoma, comprised 46.1% of the total female nonsmoking cases. In the Swedish study of Pershagen *et al.* (35), 57% of 77 female nonsmokers were adenocarcinomas and 31% squamous and small cell carcinomas. The only statistically significant ETS-associated increased risk was for squamous and small cell carcinomas, the cell types with the highest relative risks associated with active smoking. At the present time small numbers of squamous cell and small cell carcinomas in our data set preclude an adequate assessment of risk associated with ETS exposures for these cell types.

The findings of our study lend some support to the mechanism proposed by Wynder and Goodman (36) whereby inhalation of sidestream smoke might primarily increase risk of adenocarcinoma of the lung. They suggested that inhalation of sidestream smoke through the nasal passages would hinder deposition of respirable smoke particulates in the periphery of the lung while gaseous components such as volatile *N*-nitrosamines, formaldehyde, acetaldehyde, or nitrogen oxides, would be likely to reach the deeper part of the lung. Both squamous cell and small cell carcinomas tend to be centrally located, rather than in the periphery of the lung.

Our study found statistically significant elevated risks of adenocarcinoma of the lung among female nonsmokers who had had household ETS exposure or ETS exposure in occupational settings or from other sources. Each of these exposures occurred during adulthood. Exposures during the first 18 years of life were consistently unrelated to the risk of lung cancer.

Any exposure (ever/never) from a spouse who smoked was associated with at least a 30% excess risk. Increasing amount per day and years smoked significantly increased risk. The pattern of risk was the same when cases were compared to colon cancer cases or population controls and was specific for adenocarcinoma of the lung. Findings for all lung cancers combined reflect the association between ETS and adenocarcinoma of the lung diluted by the weak association with other cell types.

The internal consistency of findings with the two control groups suggests that recall bias resulting from having a diagnosis of cancer is not a likely explanation of the observed effect. The possibility remains that nonsmoking lung cancer cases and nonsmoking colon cancer cases are not similarly motivated to remember exposures to the tobacco smoke of others.

The longest duration of exposure to ETS is associated with the greatest elevation in risk, 1.43, for exposure of 30 or more years to a husbands's cigarette smoking. Although significant trends were found for other adult exposures, the dose response was not monotonic; relative risk estimates tended to decline in the longest exposure category. One possible explanation is that recall of quantitative measures of exposure is less reliable for exposures outside the home and for household members other than the spouse. A recent ten-country study was carried out by the International Agency for Research on Cancer designed to validate self-reported recent exposure of nonsmoking women to ETS from any source compared with the urinary concentration of cotinine. Duration of daily exposure to ETS from the husband was the strongest predictor of urinary cotinine (37). Studies by Pron *et al.* (38) and Coultas *et al.* (39) suggest that quantitative measures, particularly for exposures outside the home, are less reliable than categorical measures.

The lack of any association between childhood ETS exposures and lung cancer in our study, as well as the strong, consistent association with exposures during adulthood, contrasts with two recent reports by Janerich *et al.* (22) and Wu-Williams *et al.* (40). Differences in study design may contribute to the discrepant findings. About 25% ($n = 45$) of the 191 cases in the New York study were males, whereas our study was restricted to female cases ($n = 420$) (22). The authors report that there were only small differences between men and women in the amount of exposure to ETS measured by duration. The mean exposure of women to their husbands' tobacco smoke was 16.2 ± 16.7 years, while men had a mean exposure of 13.0 ± 17.0 years from smoking wives. Furthermore, there was a higher correlation between exposure from spouses lifetime ETS exposure for women in the study ($r = 0.51$) than for men ($r = 0.37$). Intensity (dose) of exposure and temporality of exposure from male and female smoker sources may differ considerably. Relatively small differences in dose, temporality, and

Table ". Association between risk* of lung cancer and childhood* exposures to tobacco smoke among nonsmoking women

Ever smoked tobacco	Cases	Colon cancer controls	Population controls	Adjusted odds ratio*	
				Colon cancer controls OR (95% CI)	Population controls OR (95% CI)
All lung carcinomas					
Father	196	189	420	0.91 (0.67–1.24)	0.82 (0.64–1.07)
Mother	44	40	97	0.85 (0.53–1.38)	0.84 (0.56–1.26)
Other household member	177	152	327	0.83 (0.59–1.18)	0.96 (0.71–1.29)
Adenocarcinoma					
Father	139	189	420	0.96 (0.69–1.35)	0.89 (0.66–1.19)
Mother	30	40	97	0.91 (0.54–1.55)	0.89 (0.56–1.43)
Other household member	125	152	327	0.81 (0.55–1.20)	0.91 (0.64–1.29)

* Adjusted for age, race, study area, annual income, and education
* Childhood is defined as first 18 years of life

duration in combination may yield more meaningful differences in exposure than that measured by duration alone. The inclusion of males in the New York study, with possibly lower doses of ETS exposure from smoking wives for fewer years and during a more recent time period, may have reduced the relative risk estimates that were not gender specific. A study in northeast China, which was comparable in size to our study, actually found a decreased risk of lung cancer associated with ETS exposures from spouses and a suggestive increased risk associated with paternal smoking (40). As suggested by the authors, these women had heavy exposures to both indoor and outdoor pollutants, which may have obscured any effect of ETS.

The studies which have examined childhood exposures are more limited than those which have focused on tobacco use by spouses, and the overall findings are inconclusive (3, 5, 11–14, 22, 41). Studies of the reliability of recall of ETS exposures suggest that recall of a parent's smoking history is less reliable than that for spouses (38, 39), and this may account in part for inconsistencies between studies. Janerich *et al.* (22) found a 2-fold increased risk associated with 25 or more smoker-years during childhood and adolescence but no increase for childhood exposures of less than 25 smoker-years (OR = 1.09). In most studies which have reported positive associations, the findings have been primarily for maternal ETS exposures in smokers rather than in nonsmokers. Correa *et al.* (5) found a significantly increased risk of lung cancer (OR = 1.36) among smokers whose mother smoked but no increased risk in nonsmokers and no elevated risk associated with the father's smoking. Wu *et al.* (14) reported a nonsignificantly elevated risk of adenocarcinoma of the lung (OR = 1.7) in females, 80% of whom had a history of smoking. Similarly, in a Swedish study of female lung cancer which included primarily smokers, a nonsignificantly elevated risk was associated with maternal (OR = 1.8) but not paternal (OR = 0.8) smoking (42). Other studies have failed to find an increased risk of lung cancer associated with childhood exposures (11, 12, 43). None of these studies examined maternal smoking as distinct from other childhood exposures. Childhood ETS exposures alone may be insufficient to increase lung cancer risk in lifetime nonsmokers but may increase risk in persons exposed transplacentally or during childhood who later smoke themselves (5).

The female lifetime nonsmokers with lung cancer in our study are considerably older than the female lung cancer cases reported in the SEER program, most of whom have actively smoked. This may represent a cohort effect; that is, older women are less likely to have smoked. The age disparity might also reflect possible differences in response among active and passive smokers. The lower dose of ETS might require a longer duration of exposure for pulmonary carcinogenesis.

Although this report represents the findings of the first 3 years of a 5-year study, it is nevertheless the largest case-control study reported to date on this topic. The findings provide additional evidence in favor of a causal relationship between exposure to ETS and lung cancer in women who have never used tobacco themselves. A dose response, not likely due to chance, was apparent for exposure to tobacco smoke during adult life from a variety of exposure sources. The association was specific for both adenocarcinoma of the lung and for all lung cancers combined compared to colon cancer.

Acknowledgments

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UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON D.C. 20460

November 20, 1992

OFFICE OF
THE ADMINISTRATOR
SCIENCE ADVISORY BOARD

EPA-SAB-IAQC-93-003

Honorable William K. Reilly
Administrator
U.S. Environmental Protection Agency
401 M Street, SW
Washington, DC 20460

Subject: Review of the Environmental Tobacco Smoke Risk Assessment - *Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders* (EPA/600/6-90/006B)

Dear Mr. Reilly:

The Science Advisory Board's (SAB) Indoor Air and Total Human Exposure Committee (IAQTHEC) met on July 21-22, 1992 to review the draft EPA document *Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders* (EPA/600/6-90/006B, May 1992 SAB Review Draft). This is the second review conducted by this committee on this issue; the first being conducted on December 4-5, 1990. The Committee reported its earlier findings to you in our April 1991 report, *An SAB Report: Review of Draft Environmental Tobacco Smoke Health Effects Document* (EPA-SAB-IAQC-91-007, April 1991). This second draft document is much revised and expanded from the first document we reviewed and, for the most part, reflects the advice presented in our April 1991 report. As a result of our recent public review, the Committee provided to the EPA Staff detailed editorial and substance comments on the draft document. The attached report contains the Committee's broad recommendations and its responses to fifteen specific questions.

The Committee is pleased to note the great improvement in the discussion and analysis of effects of environmental tobacco smoke (ETS) on the health of children. The coverage of the pertinent literature is much more complete, and the specification and quantitation of the identifiable health risks are presented in a clear and defensible manner. This new material provides a basis for the Agency to

issue an overall risk assessment on ETS that gives an appropriate emphasis to non-cancer health effects. We also note that the revised draft has an improved presentation, discussion, and analysis of ETS as a lung carcinogen, providing a firmer basis for the designation of ETS as an EPA Class A Carcinogen. The Committee was unanimous in endorsing this classification.

This second draft, as a whole, is a very substantially improved document and, in general, does a good job of presenting the scientific evidence that supports the judgment that exposure to ETS causes lung cancer and other adverse respiratory health effects. It presents the data and relevant analysis in greater detail than is available in any other single source. In general, the findings are well substantiated, although further improvements are recommended for the new Chapter 3. Other changes made from the first draft to the current draft were carefully thought out and generally appropriate. Also, the authors have done a commendable job in addressing the points raised by the Committee in its review of the first draft. This document will be an invaluable source of information for health professionals and policy makers for years to come.

We appreciate the opportunity to review this draft document and look forward to your response to our advice contained in the attached report.

Sincerely,

Raymond C. Loehr

Dr. Raymond C. Loehr, Chair
Science Advisory Board

Morton Lippmann

Dr. Morton Lippmann, Chair
Indoor Air Quality and Total
Human Exposure Committee

United States Environmental Protection Agency | Science Advisory Board A-101 Washington, DC | EPA-SAB-IAQC-93-003 November 1992

EPA

AN SAB REPORT: REVIEW OF DRAFT PASSIVE SMOKING HEALTH EFFECTS DOCUMENT

REVIEW, BY THE INDOOR AIR QUALITY AND TOTAL HUMAN EXPOSURE COMMITTEE, OF THE OFFICE OF RESEARCH AND DEVELOPMENT'S DRAFT REPORT: "RESPIRATORY HEALTH EFFECTS OF PASSIVE SMOKING: LUNG CANCER AND OTHER DISORDERS" (EPA/600/6-90/006B)

ABSTRACT

The Science Advisory Board's (SAB) Indoor Air and Total Human Exposure Committee (IAQTHEC) met on July 21-22, 1992 to review the draft EPA document *Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders* (EPA/600/6-90/006B, May 1992 SAB Review Draft). The Committee provided responses to fifteen specific questions and provided commentary on chapters of the draft document. The Committee noted the great improvement in the discussion and analysis of effects of environmental tobacco smoke (ETS) on the health of children. The coverage of the pertinent literature is much more complete in this draft than in the previous draft, and the specification and quantitation of the identifiable health risks is presented in a clear and defensible manner. This new material provides a basis for the Agency to issue an overall risk assessment on ETS that gives an appropriate emphasis to non-cancer health effects. The Committee also noted that the revised draft has an improved presentation, discussion, and analysis of ETS as a lung carcinogen, providing a firmer basis for the designation of ETS as an EPA Class A Carcinogen. The Committee was unanimous in endorsing this classification.

KEYWORDS: Environmental Tobacco Smoke; ETS; Carcinogenicity; Passive Smoking; Sidestream Smoke; Mainstream Smoke; Confounders; Lung Cancer; Respiratory Disease

NOTICE

This report has been written as a part of the activities of the Science Advisory Board, a public advisory group providing extramural scientific information and advice to the Administrator and other officials of the Environmental Protection Agency. The Board is structured to provide balanced, expert assessment of scientific matters related to problems facing the Agency. This report has not been reviewed for approval by the Agency and, hence, the contents of this report do not necessarily represent the views and policies of the Environmental Protection Agency, nor of other agencies in the Executive Branch of the Federal government, nor does mention of trade names or commercial products constitute a recommendation for use.

U.S. Environmental Protection Agency
Science Advisory Board
Indoor Air Quality and Total Human Exposure Committee

Environmental Tobacco Smoke Review

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1. EXECUTIVE SUMMARY

The Science Advisory Board's (SAB) Indoor Air and Total Human Exposure Committee (IAQTHEC) met on July 21-22, 1992 to review the draft EPA document *Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders* (EPA/600/6-90/006B, May 1992 SAB Review Draft). This is the second review conducted by this committee on this issue; the first being conducted on December 4-5, 1990. The Committee reported its findings to the EPA Administrator in our April 1991 report, *An SAB Report: Review of Draft Environmental Tobacco Smoke Health Effects Document* (EPA-SAB-IAQC-91-007, April 1991). The Committee made a number of substantive as well as structural recommendations, most of which have been addressed in the present draft document.

The Committee is pleased to note the great improvement in the discussion and analysis of effects of environmental tobacco smoke (ETS) on the health of children. The coverage of the pertinent literature is much more complete, and the specification and quantitation of the identifiable health risks are presented in a clear and defensible manner. This new material provides a basis for the Agency to issue an overall risk assessment on ETS that gives an appropriate emphasis to non-cancer health effects. We also note that the revised draft has an improved presentation, discussion, and analysis of ETS as a lung carcinogen, providing a firmer basis for the designation of ETS as an EPA Class A Carcinogen. The Committee was unanimous in endorsing this classification.

As a result of the public review, the Committee provided to the EPA Staff detailed editorial and substance comments on the draft document. This report contains the Committee's broad recommendations and its responses to fifteen specific questions in several categories from the charge to the Committee:

ETS Exposure

a) **Do the conclusions on the chemical similarities of ETS and mainstream smoke (MS) warrant the toxicological comparison between passive and active smoking made as part of the biological plausibility arguments for lung cancer (Chapter 4) and non-cancer respiratory disorders (Chapter 7)?**

Yes. All of the major carcinogenic and toxic agents found in mainstream smoke are also present in ETS, albeit at lower concentrations. The combustion of tobacco to form sidestream smoke (SS) actually results in slightly higher amounts of many toxic and carcinogenic compounds per gram of tobacco burned than when the tobacco is burned to produce mainstream smoke. However, Chapter 3, as written, did not adequately make this case and requires some modification. The following issues should be addressed in Chapter 3 to support this similarity.

- Many of the same carcinogenic compounds are found in MS and SS and ETS (measured in smoky rooms), i.e., the polycyclic aromatic hydrocarbons, the N-nitrosamines, and 4-aminobiphenyl. Many of these compounds are substantially enriched in SS relative to MS by factors ranging from about 2 to 100, although overall exposure to ETS will be much lower than to MS. Thus, as a complex mixture, SS might be expected to be somewhat more potent as a carcinogenic mixture.

- Extracts of particulate matter from combustion mixtures have consistently been shown to be carcinogenic in animal bioassays, mutagenic in short-term *in vitro* bioassays, and carcinogenic in humans, although potencies can vary.

- Many of the individual organic compounds in MS, SS and ETS have also been found to be carcinogenic in animals, e.g., many of the polycyclic aromatic hydrocarbons, the N-nitrosamines and 4-aminobiphenyl.

b) Is the extent of ETS exposure in various environments adequately characterized?

Yes. ETS exposure has been characterized in several different ways, in many environments. ETS is virtually ubiquitous, and concentrations and other characteristics have been consistently demonstrated in a very large number of environments. It is unlikely that further quantitative and qualitative description would provide much critical additional or different insights that would impact on the overall conclusions of the risk assessment.

c) Are the methods of assessing ETS exposure and the uncertainties associated with each accurately described?

Yes. The methods, and the problems and limitations are well described. Given the caveats and uncertainties, the different methods produce results which are confirmatory of each other. The questionnaire method, which has the inherent problem of information bias, has been validated, and the misclassification has been estimated in several studies. The degree of misclassification is different in different studies, as one would expect with instruments and study protocols that are different.

Lung Cancer - Hazard Identification

d) Is the evidence for the lung carcinogenicity of ETS presented adequately?

Yes. The report has been strengthened by the addition of Chapter 4 which reviews toxicologic evidence and highlights the evidence on active smoking. The epidemiologic data are adequately described, although formal criteria should be set forth for assigning the studies to tiers.

e) Does any of the new information alter the SAB conclusion regarding the categorization of ETS as an EPA Group A carcinogen?

No. The conclusion that ETS should be categorized as a Group A carcinogen, made in the initial EPA draft, remains valid with the addition of the new information in the revised draft. Despite the uncertainty about the magnitude of the risk of lung cancer due to passive smoking, the overall evidence is sufficient to declare that prolonged exposure to ETS is etiologically related to lung cancer and that ETS should be regarded as an EPA Group A carcinogen.

Lung Cancer - Population Impact

f) Is the approach used to derive estimates of U.S. female never-smoker lung cancer risk scientifically defensible?

Yes. The combination of U.S. epidemiologic studies of non-smoking women married to smokers provides an appropriate and sound basis for estimating the

relative risk of lung cancer associated with ETS among American women who have never smoked cigarettes.

g) Is the approach used to extrapolate lung cancer risk from female never-smokers to male never-smokers and former smokers of both sexes scientifically defensible?

Yes. Although alternative approaches also might have merit, the approach of extrapolating lung cancer mortality rates among women to men, which was used in the draft document, is scientifically defensible. However, the uncertainties should be further acknowledged. On the other hand, we advise caution in extending the risk assessment to ex-smokers who are known to remain at increased risk of lung cancer well beyond 5 years following cessation of smoking.

h) Are the assumptions used to derive these lung cancer-population estimates and the uncertainties involved characterized adequately?

Yes. While the overall point estimate of approximately 3,000 total lung cancer deaths (LCDs) due to ETS exposure annually in the United States is based on reasonable assumptions, the citation of a range of 2,500 to 3,300 ETS-related LCDs, based on varying only one of the parameters involved in the estimation, is misleading and implies a greater degree of precision in the estimation than is warranted. The document would be strengthened by additional acknowledgement and characterization of these uncertainties.

i) Is the degree of confidence in these estimates as stated appropriately characterized?

No. The confidence in these estimates, represented by the range of 2,500 to 3,300 deaths due to ETS, understates the uncertainties associated with each of the assumptions that went into the risk assessment. It indicates a much higher degree of precision than the 90% confidence interval surrounding the summary relative risk for spousal smoking in the U.S. of 1.19. There are other assumptions used in the risk assessment that increase the uncertainty.

Non-Cancer Respiratory Disorders

j) Have the biological plausibility arguments been adequately presented?

Yes. Biological plausibility has been adequately presented, except for the importance of bronchial responsiveness, especially in relation to lung function growth and decline.

k) Have the most important confounders been properly addressed?

Yes, most of them have. Low birth weight is a confounder that is not adequately considered by many studies. Also, ETS exposure in childhood and adolescence should be included in assessing the effects of ETS on lung functions in adults.

l) Has the weight of evidence been properly characterized? Are the conclusions scientifically defensible?

Yes. The weight of evidence has been properly characterized for most sections in Chapter 7, but the evidence for adult pulmonary function does not take into account the potential confounders of childhood respiratory diseases or exposures to parental smoking. The weight of evidence for asthma and sudden infant death syndrome (SIDS) is properly characterized, concluding causality for increased episodes and exacerbation of symptoms in asthmatics, but insufficient evidence for causality for asthma induction or SIDS.

m) Is the evidence with respect to maternal smoking and sudden infant death syndrome properly characterized? Should this evidence be included in this report?

Yes. The evidence is appropriately characterized in terms of the strong association and the Centers for Disease Control (CDC, 1991) Risk Assessment. Given the existing data, one cannot determine whether this is an *in utero* effect or an effect of environmental smoke exposure.

Population Impact

n) Is the presented population impact of ETS on lower respiratory infections and asthma in children scientifically defensible?

Yes. The issues related to asthma are clearly based on current knowledge. The focus should be on the exacerbation of asthmatic attacks because the etiology of the disease is difficult to define. The issue of childhood respiratory illnesses carries significant weight in the conclusions. It could be expanded, especially for population impact above age 18 months. Potential impact of both asthma and respiratory illnesses on adult lung function and disease could be explored further. The presentation of both a threshold and a non-threshold analysis is balanced, and the levels ascribed are consistent.

o) Are the assumptions, uncertainties, and degree of confidence in the ranges of population impact estimates adequately characterized?

Yes, for the most part. There is a complete description of the assumptions and uncertainties in the dataset. This is an enormous improvement from the first draft of this document, and provides a solid scientifically defensible base upon which to draw the conclusions made by the document.

2. INTRODUCTION

2.1 Background

The Science Advisory Board's (SAB) Indoor Air and Total Human Exposure Committee (IAQTHEC) met in public session on July 21-22, 1992 in Arlington, Virginia to review the draft EPA document *Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders* (EPA/600/6-90/006B, May 1992 SAB Review Draft). At this public meeting, the Committee received presentations from Agency staff concerning the draft document, and public comments from 15 individuals representing themselves or various groups with an interest in this matter, including the R.J. Reynolds Company, the Tobacco Institute, Action on Smoking and Health, the Coalition on Smoking OR Health, and the Centers for Disease Control. The Committee also received written comments from 27 individuals or groups. Since the Agency did not initiate a formal public comment period, the SAB was the sole recipient of formal public comments under the provisions of the Federal Advisory Committee Act. Copies of all written comments were provided to the Committee prior to the public meeting, as well as to Agency staff for their consideration in revising the draft document.

This constitutes the second review conducted by this committee on this issue. This draft document is a revision of an earlier EPA draft report formerly titled, *Health Effects of Passive Smoking: Assessment of Lung Cancer in Adults and Respiratory Disorders in Children*, which the Committee reviewed on December 4 and 5, 1990. Our earlier findings (*An SAB Report: Review of Draft Environmental Tobacco Smoke Health Effects Document*, EPA-SAB-IAQC-91-007, April 1991) concurred with EPA's conclusions that ETS should be designated a Group A or known human carcinogen, but suggested that the conclusions on respiratory disorders in children could be made stronger. Our report also suggested several areas in which the health risk assessment could be improved, and offered to provide additional advice on a revised document.

The draft document has been significantly revised from the 1990 draft, and is quite different in size as well as format. The current draft is about 600 pages in length, compared to the 350 of the earlier draft. The increased size is the result of several changes, including: a new Chapter (3) on physical/chemical components and assessing exposure to ETS; a new Chapter (4) on the relationship of active smoking

and lung cancer; an expanded Appendix (Appendix A), which includes a review of all the pertinent ETS lung cancer studies in non-smoking women; a rewrite of the noncancer respiratory disorders Chapter (7) to include approximately thirty more studies than did the 1990 draft; and a new quantitative risk assessment Chapter (8) on noncancer respiratory effects.

As a result of our earlier review, two appendices from the EPA initial draft were dropped in the revision. These dealt mostly with lung deposition modelling and active to passive smoking dose-response modelling. As we suggested in our earlier report, some of the material from these appendices was extracted and included in Chapters 3 and 4 of the revised draft.

2.2 Charge to the Committee

The Agency sought the advice of the SAB on the accuracy and completeness of the entire document and on whether the Agency has been responsive to the Committee's previous recommendations. In addition, the SAB was asked to address the following specific issues:

A - ETS EXPOSURE (Chapter 3)

a) Do the conclusions on the chemical similarities of ETS and MS warrant the toxicological comparison between passive and active smoking made as part of the biological plausibility arguments for lung cancer (Chapter 4) and non-cancer respiratory disorders (Chapter 7)?

b) Is the extent of ETS exposure in various environments adequately characterized?

c) Are the methods of assessing ETS exposure and the uncertainties associated with each accurately described?

B - LUNG CANCER

1. HAZARD IDENTIFICATION (Chapters 4 and 5)

   a) Is the evidence for the lung carcinogenicity of ETS presented adequately?

   b) Does any of the new information alter the SAB conclusion regarding the categorization of ETS as an EPA Group A carcinogen?

2. POPULATION IMPACT (Chapter 6)

   a) Is the approach used to derive estimates of U.S. female never-smoker lung cancer risk scientifically defensible?

   b) Is the approach used to extrapolate lung cancer risk from female never-smokers to male never-smokers and former smoker of both sexes scientifically defensible?

   c) Are the assumptions used to derive these lung cancer population estimates and the uncertainties involved characterized adequately?

   d) Is the degree of confidence in these estimates as stated appropriately characterized?

C - NON-CANCER RESPIRATORY DISORDERS

1. HAZARD IDENTIFICATION (Chapter 7; Sections 8.1 and 8.2)

   a) Have the biological plausibility arguments been adequately presented?

   b) Have the most important confounders been properly addressed?

c) Has the weight of evidence been properly characterized? Are the conclusions scientifically defensible?

d) Is the evidence with respect to maternal smoking and sudden infant death syndrome properly characterized? Should this evidence be included in this report?

2. POPULATION IMPACT (Chapter 8)

a) Is the presented population impact of ETS on lower respiratory infections and asthma in children scientifically defensible?

b) Are the assumptions, uncertainties, and degree of confidence in the ranges of population impact estimates adequately characterized?

2.3 Format of this report

Although a major focus of the Committee's review is the response to the questions in the charge, the Committee also provided detailed commentary on each chapter of the document, both in the meeting transcript and in written comments. This report consists of 4 major chapters. An Executive Summary, an Introduction, Responses to the Questions in the Charge, and Comments on the Draft Document. Detailed editorial comments were omitted.

3. RESPONSES TO THE QUESTIONS IN THE CHARGE

3.1 Questions Concerning ETS Exposure (Chapter 3)

a) Do the conclusions on the chemical similarities of ETS and mainstream smoke (MS) warrant the toxicological comparison between passive and active smoking made as part of the biological plausibility arguments for lung cancer (Chapter 4) and non-cancer respiratory disorders (Chapter 7)?

Yes, all of the major carcinogenic and toxic agents found in MS are also present in ETS, albeit at lower concentrations. The combustion of tobacco to form ETS actually results in slightly higher amounts of many toxic and carcinogenic compounds per gram of tobacco burned than when the tobacco is burned to produce mainstream smoke. There are substantial differences in the relative composition of the smoke formed between mainstream and sidestream smoke, similar to the differences between the MS formed by different currently manufactured cigarettes, but there is no reason to suppose that the qualitative toxicities of ETS and MS are substantively different. In comparing these two agents the differences are largely ones of dose and duration of exposure rather than fundamental differences in the toxicity or carcinogenicity of the agent in question. However, Chapter 3, as written, did not adequately make this case and requires some modification. The following issues should be addressed in Chapter 3 to support this similarity.

- Many of the same carcinogenic compounds are found in MS and SS and ETS (measured in smoky rooms), i.e., the polycyclic aromatic hydrocarbons, the N-nitrosamines, and 4-aminobiphenyl. Many of these compounds are substantially enriched in SS relative to MS by factors ranging from about 2 to 100, although overall exposure to ETS will be much lower than to MS. Thus, as a complex mixture, SS might be expected to be somewhat more potent as a carcinogenic mixture.

- Extracts of particulate matter from combustion mixtures have consistently been shown to be carcinogenic in animal bioassays, mutagenic in short-term *in vitro* bioassays, and carcinogenic in humans, although potencies can vary.

- Many of the individual organic compounds in MS, SS and ETS have also been found to be carcinogenic in animals, e.g., many of the polycyclic aromatic hydrocarbons, the N-nitrosamines and 4-aminobiphenyl.

b) Is the extent of ETS exposure in various environments adequately characterized?

Adequate is a relative word. ETS exposure has been characterized in several different ways, in many environments. ETS is virtually ubiquitous, and concentrations and other characteristics have been consistently demonstrated in a very large number of environments. It is unlikely that further quantitative and qualitative description would provide much critical additional or different insight that would impact on the overall conclusions of the risk assessment.

Precise calculation of the expected risk due to ETS exposure would require detailed estimates on a population base for all of the United States for various ETS exposures. This database does not exist. However, there is a vast array of data on samples of the population and samples of the environments that can be extrapolated to the U.S. general population with an acceptable degree of certainty. These measures include traditional atmospheric measures of constituents of the smoke as well as individual dosimetry from individual monitors and from samples of biologic fluids for constituents of tobacco smoke and/or metabolic products of such constituents. As the report describes, one would expect some variation in the U.S. population in the degree of exposure to these agents, and the report describes the process for taking into account this variability in calculating numbers of deaths that are presented in the document. The data on exposure to the population in question are adequate to make the risk assessments that have been made, and are substantially more complete than the exposure estimates that are available for many other Class A carcinogens.

c) Are the methods of assessing ETS exposure and the uncertainties associated with each accurately described?

The methods, and the problems and limitations are well described. Given the caveats and uncertainties, the different methods produce results which are confirmatory of each other. The questionnaire method, which has inherent problems of information bias, has been validated, and the misclassification has been

estimated in several studies. The degree of misclassification is different in different studies, as one would expect with instruments and study protocols that are different.

Although several possible markers for ETS are mentioned, two, respirable suspended particulate matter (RSP) and nicotine, are discussed most fully. The major uncertainties related to RSP as a marker are that the other sources of RSP in all environments vary from environment to environment. At low smoking levels, the RSP attributable to ETS is difficult to determine directly, and estimates of ETS in these cases are unstable. By contrast, ETS is the only source of nicotine in most environments, so that even at low smoking levels nicotine can be quantified and attributed entirely to ETS. The major limitations of vapor phase nicotine are that it is deposited onto surfaces more quickly than other components of ETS and also is re-emitted from these surfaces. On the other hand, the successful use of these two markers in field studies argues that these uncertainties are relatively small. The wide range of ETS concentrations reported in human environments where smoking occurs--over two and up to three orders of magnitude--indicate that the current degree of uncertainty in assessing ETS exposures can be tolerated.

Of the possible biomarkers of ETS exposure, cotinine and nicotine in body fluids (saliva, blood, and urine) are discussed most extensively. The advantages and limitations of these are accurately described, except that there should be some discussion of the possible dietary sources of nicotine and cotinine, and the contribution these might make to measured levels of these compounds in nonsmokers.

3.2 Questions Concerning Lung Cancer - Hazard Identification (Chapters 4 and 5)

a) Is the evidence for the lung carcinogenicity of ETS presented adequately?

Yes, the report has been strengthened by the addition of Chapter 4 which reviews toxicologic evidence and highlights the evidence on active smoking. The epidemiologic data are adequately described, although formal criteria should be set forth for assigning the studies to tiers.

The evidence for the carcinogenicity of ETS is presented in several logical components: evidence for MS inhalation being causal for lung cancer in proportion to dose, evidence for the reduction in risk following cessation of smoking, animal evidence for the carcinogenicity of MS and ETS, and evidence of mutagenicity of MS end ETS components. Finally the results of a large number of epidemiological studies of ETS exposures in non-smoking and never-smoking populations, and the increase in lung cancer risk associated with such exposures, are presented in great detail. These community exposures are shared by a very large fraction of the population and, although the risks are small compared to the risks of active smoking, they are quite consistent. Any studies involving low relative risks are likely to be controversial: there always are methodological short-comings.

b) Does any of the new information alter the SAB conclusion regarding the categorization of ETS as an EPA Group A carcinogen?

The conclusion that ETS should be categorized as a Group A carcinogen, made in the initial EPA draft, remains valid with the addition of the new information in the revised draft. The totality of epidemiologic evidence indicates that non-smoking women married to smokers have experienced an increased risk of lung cancer. Increased risks have been observed in most of the investigations in various parts of the world, including the United States. Biomarker studies have shown that nonsmokers exposed to ETS inhale and metabolize compounds in cigarette smoke, leaving no doubt that some of the various classes of carcinogens in tobacco smoke reach bodily tissues. Although concentrations of tobacco smoke components are much lower in ETS than in mainstream cigarette smoke, ETS and MS share many qualitative chemical similarities and, for many carcinogens, the relative proportion in ETS is enriched. Because cigarette smoking is such a well-established risk factor for lung cancer, and one that exhibits no evidence for a threshold, it is biologically plausible that prolonged inhalation of ETS results in some increase in risk of lung cancer among nonsmokers. Thus, despite the uncertainty about the magnitude of the risk of lung cancer due to passive smoking, the overall evidence is sufficient to declare that prolonged exposure to ETS is etiologically related to lung cancer and that ETS should be regarded as an EPA Group A carcinogen.

3.3 Questions Concerning Lung Cancer - Population Impact (Chapter 6)

a) Is the approach used to derive estimates of U.S. female never-smoker lung cancer risk scientifically defensible?

The combination of U.S. epidemiologic studies of non-smoking women married to smokers provides an appropriate and sound basis for estimating the relative risk of lung cancer associated with ETS among American women who have never smoked cigarettes. The procedures used to estimate the numbers of lung cancer deaths due to ETS among female nonsmokers in the United States are based on a series of reasonable assumptions, and yield a point estimate that is scientifically defensible.

b) Is the approach used to extrapolate lung cancer risk from female never-smokers to male never-smokers and former smokers of both sexes scientifically defensible?

The use of data among non-smoking women to estimate lung cancer deaths (LCDs) due to ETS among non-smoking men offers the advantage of the much greater stability of the estimated effect of ETS among non-smoking women. The epidemiologic studies in the United States have included over 1,000 non-smoking women with lung cancer, but the numbers of non-smoking male cases studied are nearly an order of magnitude smaller, and probably too few to provide an adequate basis for national projections of ETS-related LCDs. Information to make direct estimates of ETS effects among former smokers is even more limited and should take into account their residual risk. Thus, although alternative approaches also might have merit, the approach of extrapolating lung cancer mortality rates among women to men, which was used in the draft document, is scientifically defensible. However, the uncertainties should be further acknowledged. On the other hand, we advise caution in extending the risk assessment to ex-smokers who are known to remain at increased risk of lung cancer well beyond 5 years following cessation of smoking.

c) Are the assumptions used to derive these lung cancer population estimates and the uncertainties involved characterized adequately?

The estimated numbers of lung cancer deaths (LCDs) due to ETS in the United States are based upon several assumptions. For never-smoking women, the

numbers rely on estimates of the relative risk of lung cancer among non-smoking women married to smokers vs. nonsmokers, the effect of potential misclassification of ETS exposure, the relative increase above background in ETS exposure among non-smoking women married to smokers, the proportions of women in various marital/ETS/smoking exposure categories, and the relative risk of lung cancer among female active smokers. Assumptions associated with procedures to derive these estimates are provided, but the draft document does not adequately convey the extent of uncertainty in the resulting estimated numbers of ETS-induced LCDs among never-smoking women. Uncertainties for the estimates among never-smoking males or former smokers are even greater. While the overall point estimate of approximately 3,000 total LCDs due to ETS exposure annually in the United States is based on reasonable assumptions, the citation of a range of 2,500 to 3,300 ETS-related LCDs, based on varying only one of the parameters involved in the estimation, is misleading and implies a greater degree of precision in the estimation than is warranted. The document would be strengthened by additional acknowledgement and characterization of these uncertainties.

d) Is the degree of confidence in these estimates as stated appropriately characterized?

The confidence in these estimates, represented by the range of 2,500 to 3,300 deaths due to ETS, understates the uncertainties associated with each of the assumptions that went into the risk assessment. It indicates a much higher degree of precision than the 90% confidence interval surrounding the summary relative risk for spousal smoking in the U.S. of 1.19. There are other assumptions used in the risk assessment that increase the uncertainty.

3.4 Questions Concerning Noncancer Respiratory Disorders - Hazard Identification (Chapter 7; Sections 8.1 and 8.2)

a) Have the biological plausibility arguments been adequately presented?

Biological plausibility has been adequately presented, except for the importance of bronchial responsiveness, especially in relation to lung function growth and decline.

b) Have the most important confounders been properly addressed?

Most of them have. Low birth weight is a confounder that is not adequately considered by many studies. Also, ETS exposure in childhood and adolescence should be included in assessing the effects of ETS on lung function in adults.

c) Has the weight of evidence been properly characterized? Are the conclusions scientifically defensible?

Yes. The weight of evidence has been properly characterized for most sections in Chapter 7, but the evidence for adult pulmonary function does not take into account the potential confounders of childhood respiratory diseases or exposures to parental smoking. The weight of evidence for asthma and sudden infant death syndrome (SIDS) is properly characterized, concluding causality for increased episodes and exacerbation of symptoms in asthmatics, but insufficient evidence for causality for asthma induction or SIDS.

d) Is the evidence with respect to maternal smoking and sudden infant death syndrome properly characterized? Should this evidence be included in this report?

The evidence is appropriately characterized in terms of the strong association and the suggestion that it is impossible, given the existing data, to determine whether this is an *in utero* effect or an effect of environmental smoke exposure.

3.5 Questions Concerning Noncancer Respiratory Disorders - Population Impact (Chapter 8)

a) Is the presented population impact of ETS on lower respiratory infections and asthma in children scientifically defensible?

Yes. The issues related to asthma are clearly discussed based on current knowledge. The focus should be on the exacerbation of asthmatic attacks because the etiology of the disease is difficult to define. The issue of childhood respiratory illnesses carries significant weight in the conclusions. It could be expanded, especially for population impact above age 18 months. Potential impact of both asthma and respiratory illnesses on adult lung function and disease could be explored further. The presentation of both a threshold and a non-threshold analysis is balanced, and the levels ascribed are consistent.

b) Are the assumptions, uncertainties, and degree of confidence in the ranges of population impact estimates adequately characterized?

Yes, there is a complete description of the assumptions and uncertainties in the data set. This is a enormous improvement from the first draft of this document, and provides a solid scientifically defensible base upon which to form the conclusions drawn by the document.

4. COMMENTS ON THE DRAFT DOCUMENT

This second draft as a whole, is a very substantially improved document and, in general, does a good job of presenting the scientific evidence that supports the judgment that exposure to ETS causes lung cancer and other adverse respiratory health effects. It presents the data and relevant analysis in greater detail than is available in any other single source. The findings in general are well substantiated. The changes made from the first draft to the current draft were carefully thought out and generally appropriate. Also, the authors have done a commendable job in addressing the points raised by the Committee in its review of the first draft. This document will be an invaluable source of information for health professionals and policy makers for years to come.

In preparing the final draft, the Committee recommends that EPA consider a change in organization that we believe would better clarify the nature of the report and its findings. We suggest that Chapters 4, 5 and 6 cover the health effects of ETS on adults, and Chapters 7 and 8 cover the health effects of children rather than having Chapters 4-6 devoted to cancer and Chapters 7 and 8 devoted to other disorders.

4.1 Chapter 1 - Summary and Conclusions

The summary and conclusions of the report are clearly described with a brief and appropriate discussion of substantiating evidence. Both the strengths and limitations of the data on which conclusions are based are described. The addition of comments on the relative strengths of the conclusions is also helpful. This chapter will be very useful to the reader who does not have time or the expertise to read the entire document.

A problem with this chapter is that although the findings and conclusions are accurately reported from the document, summary comments and conclusion comments are not clearly separated. The authors may wish to examine summary and conclusions chapters from Surgeon General's reports (DHHS, 1984; 1986) to give them an idea how better to organize this material. Clearly these two should be separated. It would also be helpful to have each summary statement stand alone, be in paragraph form, or be highlighted. In addition, it would also be

helpful if some clearly stated overall objective for the document and specific objectives for each chapter were outlined.

Because of the importance of this Chapter, the conclusions should be clearly highlighted, or a new chapter on Conclusions and Recommendations should be added. At a minimum, we recommend that a new section: 1.3 Conclusions be added in which the conclusions reached as a result of analyses of findings are summarized.

4.2 Chapter 2 - Introduction

This chapter does provide a brief statement of the problem of health risks related to tobacco use. This introductory chapter should clearly establish why ETS is an important problem. The terms "passive smoking," "ETS," and "*in utero* vs. post-natal exposure" need to be more clearly defined. Specifically, it is important to distinguish between passive smoking and ETS. Chapter 2 suffers from excessive use of epidemiologic statistical and risk assessment jargon. It is critical for the general understanding and acceptance of this EPA report that clear language be presented in these chapters to make complex scientific issues accessible and understandable to a lay audience.

4.3 Chapter 3 - Estimation of Environmental Tobacco Smoke Exposure

This chapter does a good job of covering the included material. However, there are some important points related to the estimation of exposure to ETS that are not included in the review. These points are important because they affect the interpretation of the exposure and risk estimates.

The introduction to the chapter correctly points out the various factors that can affect the composition of ETS including the variability of emission rates, frequency of smoking, types of products combusted, other sources of the measured components, ventilation rates and effects due to chemistry and deposition losses. However, the review then treats ETS as a material having constant composition with little regard to some of these factors. Three points with respect to the chemical composition of ETS in indoor environments seem especially important.

4.3.1 The composition of toxic compounds in ETS

The review should point to the expectation that the toxic potential of a unit mass of ETS is expected to be greater than that of MS. This discussion might also include some comments on the known changes in the chemical composition of ETS as it ages and the unknown impact these changes have on toxicology.

4.3.2 The rate of removal of nicotine in indoor environments

Nicotine has been the component of choice as a tracer of ETS in many studies of environmental tobacco smoke because of its uniqueness to ETS and ease of measurement. However, it has also been shown by several investigators that nicotine is the component of ETS that is most rapidly removed in indoor environments. The end result of its removal will be the underestimation of exposure to most other components of ETS when the estimate is based on the concentration of nicotine present in an indoor environment, with the underestimation becoming more severe as the exposure approaches zero. Underestimation will have the same effect as random misclassification, driving the data towards the null hypothesis. This effect should not be ignored.

4.3.3 The relative amounts of ETS and non-ETS particles in indoor environments

The text frequently emphasizes the average excess of ETS particulate matter seen in indoor environments where smoking is present, but does not put these numbers into proper context. The impression given is that the bulk of particulate matter in typical indoor environments is from ETS, with high concentrations being rarely measured in the absence of smoking. The average contribution, however, is that about half of the fine particulate matter in a typical indoor environment where smoking is present comes from ETS.

4.4 Chapter 4 - Hazard Identification I: Lung Cancer in Active Smokers, Long-term Animal Bioassays, and Genotoxic Studies

This chapter provides a concise and adequate summary of relevant but indirect evidence supporting the biological plausibility for a causal association between long-term exposure to ETS and an elevated risk for lung cancer. The clear dose-related association of lung cancer risk with exposure to MS smoke, the

presence of essentially all of the same known carcinogens in both MS and SS smoke, the laryngeal cancers produced by inhaled MS smoke in the Syrian hamster, and the greater carcinogenic potency of SS tar than MS tar in the mouse skin, all are consistent with an elevated risk of lung cancer in nonsmokers chronically exposed to ETS.

4.5 Chapter 5 - Hazard Identification II: Interpretation of Epidemiologic Studies on ETS and Lung Cancer

This chapter provides an overview of 31 epidemiologic studies of ETS and lung cancer, an evaluation of these studies, and a meta-analysis. Summary analyses are presented by geographic region and study quality. The chapter ends with an overall evaluation of the evidence and a conclusion that ETS is a Group A human carcinogen. We support the general approach of reviewing the studies, classifying them by quality, and calculating a pooled measure of association. However, a number of specific problems should be addressed.

a) Lack of a general epidemiologic framework: The chapter fails to set out a general framework for evaluating the studies. The introductory portions of the chapter, in addition to considering the issue of statistical significance, should systematically review the types of bias that may affect case-control and cohort studies. The framework could then be used for evaluating the individual studies. The types of relevant bias can be readily listed: selection bias, information bias resulting in either differential or non-differential misclassification, and confounding. Improper control group selection and statistical power are two additional limitations. We suggest an overall discussion of each of these methodological limitations and the potential consequences for interpreting the studies. This approach would lend itself to evaluating the individual studies.

b) Lack of framework for reviewing individual studies: A second concern, failure to develop a systematic framework for reviewing the individual studies, would be satisfactorily addressed by the approach suggested above.

c) Editing: The text needs further editing. In many instances epidemiologic terminology has been used inappropriately. This imprecision of language may lead to unneeded ambiguity.

d) Confounding: A substantial portion of the text is directed at potential bias introduced by confounding. As noted by the authors, confounding variables are themselves risk factors but are also associated with the exposure of interest. We presently know little about causes of lung cancer in persons who have never smoked. Some occupational factors are causally linked to lung cancer in never smokers but other risk factors have not yet been well established. For example, while diet is emphasized as a potential confounder in the chapter, we are still awaiting confirmation that dietary factors are of importance in the high proportion of lung cases occurring in smokers.

4.6 Chapter 6 - Population Risk of Lung Cancer from Passive Smoking

Chapter 6 provides estimates of the numbers of lung cancer deaths (LCD) among nonsmokers in the United States attributed to ETS exposure. Estimates are presented first for female nonsmokers, the group with the largest quantity of relevant data on the effects of ETS. The estimated number of LCDs depends upon the estimate, presented and discussed in Chapter 5, of 1.19 for the relative risk of lung cancer among American non-smoking women married to smokers compared to non-smoking women married to nonsmokers, and the relative risk estimate of 1.59 for these same women corrected for background ETS exposure. Based on these two estimates, and estimates of the relative risk of lung cancer among female smokers compared to nonsmokers, the proportions of non-smoking women exposed to spousal ETS, and the proportion of women who are nonsmokers, the report partitions the approximately 38,000 LCDs in American women in 1985 into about 32,000 due to active smoking, 1,500 due to ETS (1,030 due to background ETS and 470 due to spousal ETS), and about 5,000 due to other causes. No confidence limits are placed about the 1,500 ETS-related LCDs to take into account inherent sampling variation. The report then estimates the numbers of LCDs in 1985 attributed to ETS among male nonsmokers. Limited data on relative risks of lung cancer among non-smoking men married to smoking women are available from 11 studies (7 in the United States), with an overall average relative risk of about 1.6 (1.4 for U.S. studies), but the report does not use these estimates to estimate LCDs attributed to ETS among men. Instead it extrapolates from the more stable

estimated lung cancer mortality rates among non-smoking women married to smokers vs. nonsmokers. So doing yields an estimate of 500 LCDs due to ETS among male nonsmokers.

A similar extrapolation technique is used to estimate the number of LCDs in 1985 due to ETS among male and female former smokers, where former smokers are defined as those who quit smoking 5 or more years ago. The estimated number of ETS-related LCDs among these former smokers is 1,060. The grand total of LCDs attributed to ETS is thus 3,060.

Data from the large, multi-center case-control study of lung cancer among non-smoking women by Fontham et al. (1991) are then used in place of the combined U.S. data to produce alternative total estimates of 2,480 and 3,300 ETS--induced LCDs. The two estimates from this case-control study arise from use of two different estimates (one based on means, the other based on medians) of the ratio of ETS exposure in non-smoking women married to smokers vs. nonsmokers.

The use of data only from American studies to estimate LCDs in the United States seems justifiable. Data from other countries bolster the conclusion that ETS exposure can increase risk of lung cancer in humans (i.e., that ETS is a class A carcinogen), but the magnitude of the effect in the United States seems better estimable using the U.S. data alone. The use of data among non-smoking women to estimate LCDs due to ETS among non-smoking men offers the advantage of the much greater stability of the estimated effect of ETS among non-smoking women. The U.S. studies included over 1,000 non-smoking women with lung cancer, but the number of non-smoking male cases studied appears to be nearly an order of magnitude smaller, and probably too few to provide an adequate basis for national projections of ETS-related LCDs. The chapter cites problems in adjustment for misclassification as the rationale for not using the male data, but a stronger case might be made on grounds of limited information among males and the considerably greater precision of the female data. Nevertheless, the text should more fully discuss the claim that misclassification bias may be greater for male than female nonsmokers, and acknowledge the possibility that the number of ETS--related LCDs among male nonsmokers may be less than, in addition to greater than, the estimate of 500.

The decision to not use cigarette-equivalent low-dose interpolation techniques to estimate the numbers of LCDs due to ETS seems appropriate. It is not clear

which one or several of the many carcinogens in cigarette smoke is responsible for the increased risk of lung cancer in active smokers, nor how variation in bodily cotinine concentration relates to variation in lung exposure to these carcinogens. Thus, there would be considerable uncertainty in using cotinine or other biomarker data to translate ETS exposure by nonsmokers into cigarette-equivalent exposures. The biomarker data clearly show that nonsmokers inhale and metabolize components of tobacco smoke, and help establish that exposure to ETS can increase the risk of cancer, but they are less useful in estimating the number of cancers induced by ETS.

The type of risk estimation done in this section is always subject to error because of inadequate knowledge in many of the parameters and the need to make various assumptions about the relationship of these parameters to one another. The estimates obtained in this section appear reasonable and the assumptions are clearly stated. Analytically it would seem difficult to place confidence intervals that can be interpreted in the conventional context of a sample size. Some indication in this regard could be accomplished with a simulation study. The sensitivity analysis serves to convey the high degree of variability in the estimates but considers primarily one variable at a time. The approach used to estimate lung cancer due to ETS in non-smoking women appears reasonable although the parameters used are presently based on untestable assumptions. The extrapolation to males and ex-smokers requires additional assumptions and, thus, is more difficult to defend scientifically. However, the report states the assumptions made to obtain the estimates.

4.7 Chapter 7 - Passive Smoking and Respiratory Disorders Other Than Cancer

This chapter is an important component to the report. This chapter is substantially improved from the previous draft. Section 7.2.2 presents evidence that the *in utero* exposure to cigarette smoke via the mother significantly affects neonatal pulmonary function both in animals and humans. While the evidence is fragmentary, the effects are consistent and convincing overall.

Section 7.2.3 indicates that reduced airway size and reduced respiratory flow rates in neonates are associated with an increased of risk of pulmonary infection during infancy, and possibly a predisposition to chronic obstructive lung disease in adulthood. The section provides an important theoretical link between *in utero* or

early life exposure to cigarette smoke and possible abnormal lung function and pulmonary morbidity in later life.

Sections 7.2.4 and 7.2.5. provide evidence that parental smoking, at least heavy smoking, enhances the likelihood of bronchial hyper-responsiveness and other atopic illnesses in children. These findings are even more striking in light of the "healthy smoker effect". Overall, Section 7.2 provides an excellent overview of biological mechanisms that may underlie the ETS-respiratory disease association.

New data reviewed in Section 7.3 support prior conclusions of the Surgeon General's Report (DHHS, 1986) and the National Research Council (NRC, 1986) report that parental smoking is associated with increased risk of respiratory tract infections in infants. The Chen (1989), Chen et al. (1986; 1988) and Woodward et al. (1990) data demonstrating that *in utero* exposure via maternal cigarette smoking is not the sole explanation for the increased risk represent a valuable new observation. Several potential confounders were discussed and convincingly argued not to account for the observed risk of ETS exposure; these included low birth weight. Low birth weight, the risk of which has been reported to be increased by ETS exposure, would be expected to raise the risk of respiratory infection during infancy.

Section 7.5. The recent studies on cough, phlegm or wheezing and ETS are particularly impressive in that so many studies performed in different countries and with very different methodologies have arrived at similar conclusions. Potential confounders are thoroughly discussed. The conclusion that ETS exposure is associated with an increased prevalence of respiratory symptoms in infants and the young is highly reasonable.

Section 7.6. The new studies in this area offer support for the conclusion that ETS exposure aggravates asthma in children. The discussion that it is more likely chronic rather than acute exposure to ETS that increases airway responsiveness, the importance of high levels of exposure (which may vary seasonally), and the socioeconomic influences on exposure are important for our understanding of the nature of the relationship between ETS and asthma, as well as for developing prevention strategies.

Section 7.7. The studies on maternal smoking and risk of sudden infant death syndrome (SIDS) and the Centers for Disease Control (CDC, 1991) Risk

Assessment are remarkably consistent in showing an increased risk, even though patients were studied in different areas in the U.S. and in different countries around the world. The association between maternal smoking and SIDS persists after controlling for a variety of potential confounders, including low birth weight. However, the relative risks of prenatal and postnatal cigarette smoke exposure cannot be distinguished.

Section 7.8. As noted, the data on ETS and pulmonary health in adults are inconsistent from study to study and potentially confounded by exposure to other respiratory irritants that are linked to socioeconomic class and/or to a greater tolerance by ETS-exposed individuals of other noxious airborne chemicals. Not mentioned in the discussion of potential confounders is the issue of childhood exposure that could influence pulmonary function in adulthood. It is likely that children whose parents smoked, even if they did not become smokers themselves, are more likely to marry a smoker compared to children whose parents do not smoke. Also relevant to the issue is the study of Janerich et al. (1990) indicating that a substantial fraction of life-long environmental ETS exposure occurs during childhood and adolescence.

4.8 Chapter 8 - Assessment of Increased Risk for Respiratory Illnesses in Children from Environmental Tobacco Smoke

In general, the risk assessments in this chapter are well done, and tend to be on the conservative side. For one thing, there is a tendency toward an underestimation of the risk associated with the use of cotinine as a measure of exposure to ETS. For another, the risk assessment was limited, for LRI's to those under age 18 months, for asthmatic attacks on children and only those exposed at home and requiring some type of urgent care; it did not include the risk associated with bronchial responsiveness nor the risk associated with ETS-induced respiratory effects in childhood which have high probabilities of producing/impacting on adult respiratory problems.

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Current Intelligence Bulletin 54

June 1991

ENVIRONMENTAL TOBACCO SMOKE IN THE WORKPLACE

Lung Cancer and Other Health Effects

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Centers for Disease Control
National Institute for Occupational Safety and Health

CDC
CENTERS FOR DISEASE CONTROL

CURRENT INTELLIGENCE BULLETIN 54

ENVIRONMENTAL TOBACCO SMOKE IN THE WORKPLACE

Lung Cancer and Other Health Effects

U.S. Department of Health and Human Services
Public Health Service
Centers for Disease Control
National Institute for Occupational Safety and Health
Division of Standards Development and Technology Transfer
Division of Surveillance, Hazard Evaluations, and Field Studies

June 1991

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FOREWORD

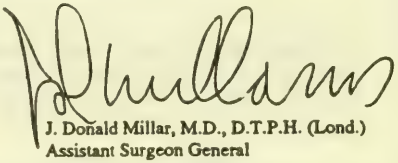
Current Intelligence Bulletins (CIBs) are issued by the National Institute for Occupational Safety and Health (NIOSH), Centers for Disease Control, Atlanta, Georgia, to disseminate new scientific information about occupational hazards. A CIB may draw attention to a formerly unrecognized hazard, report new data on a known hazard, or disseminate information on hazard control.

CIBs are distributed to representatives of academia, industry, organized labor, public health agencies, and public interest groups, as well as to Federal agencies responsible for ensuring the safety and health of workers.

Copies are available to individuals upon request from the Division of Standards Development and Technology Transfer, NIOSH (Robert A. Taft Laboratories, 4676 Columbia Parkway, Cincinnati, Ohio 45226). We welcome suggestions concerning the content, style, and distribution of these documents.

The purpose of this bulletin is to disseminate information about the potential risk of cancer to workers exposed to environmental tobacco smoke (ETS). In 1964, the Surgeon General issued the first report on smoking and health, which concluded that cigarette smoking causes lung cancer. Since then, research on the toxicity and carcinogenicity of tobacco smoke has demonstrated that the health risk from inhaling tobacco smoke is not limited to the smoker, but also includes those who inhale ETS. ETS contains many of the toxic agents and carcinogens that are present in mainstream smoke, but in diluted form. Recent epidemiologic studies support and reinforce earlier published reviews by the Surgeon General and the National Research Council demonstrating that exposure to ETS can cause lung cancer. These reviews estimated the relative risk of lung cancer to be approximately 1.3 for a nonsmoker living with a smoker compared with a nonsmoker living with a nonsmoker. In addition, recent evidence also suggests a possible association between exposure to ETS and an increased risk of heart disease in nonsmokers. Although these data were not gathered in an occupational setting, ETS meets the criteria of the Occupational Safety and Health Administration (OSHA) for classification as a potential occupational carcinogen [Title 29 of the *Code of Federal Regulations*, Part 1990]. NIOSH therefore considers ETS to be a potential occupational carcinogen and recommends that exposures be reduced to the lowest feasible concentration. All available preventive measures should be used to minimize occupational exposure to ETS.

NIOSH urges employers to disseminate this information to employees. NIOSH also requests that professional and trade associations and unions inform their members about the potential hazards of exposure to ETS. Readers seeking more detailed information about the studies cited in this bulletin are urged to consult the original publications.



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ABSTRACT

The National Institute for Occupational Safety and Health (NIOSH) has determined that environmental tobacco smoke (ETS) is potentially carcinogenic to occupationally exposed workers. In 1964, the Surgeon General issued the first report on smoking and health, which concluded that cigarette smoke causes lung cancer. Since then, research on the toxicity and carcinogenicity of tobacco smoke has demonstrated that the health risk from inhaling tobacco smoke is not limited to the smoker, but also includes those who inhale ETS. ETS contains many of the toxic agents and carcinogens that are present in mainstream smoke, but in diluted form. Recent epidemiologic studies support and reinforce earlier published reviews by the Surgeon General and the National Research Council demonstrating that exposure to ETS can cause lung cancer. These reviews estimated the relative risk of lung cancer to be approximately 1.3 for a nonsmoker living with a smoker compared with a nonsmoker living with a nonsmoker. In addition, recent evidence suggests a possible association between exposure of nonsmokers to ETS and an increased risk of heart disease.

Although these data were not gathered in an occupational setting, ETS meets the criteria of the Occupational Safety and Health Administration (OSHA) for classifying substances as potential occupational carcinogens [Title 29 of the *Code of Federal Regulations*, Part 1990]. NIOSH therefore recommends that ETS be regarded as a potential occupational carcinogen in conformance with the OSHA carcinogen policy, and that exposures to ETS be reduced to the lowest feasible concentration. Employers should minimize occupational exposure to ETS by using all available preventive measures.

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ABBREVIATIONS

ASPH	Association of Schools of Public Health
C	carcinogenic
CO	carbon monoxide
COHb	carboxyhemoglobin
CoC	cocarcinogenic
CI	confidence interval
CIB	Current Intelligence Bulletin
°C	degree Celsius
DSDTT	Division of Standards Development and Technology Transfer
DSHEFS	Division of Surveillance, Hazard Evaluations, and Field Studies
EPA	U.S. Environmental Protection Agency
ETS	environmental tobacco smoke (tobacco smoke in the ambient atmosphere composed of sidestream smoke and exhaled mainstream smoke)
IARC	International Agency for Research on Cancer
m	meter
MS	mainstream smoke (smoke drawn through the tobacco and into the smoker's mouth)
mg	milligram
ml	milliliter
mm	millimeter
MSHA	Mine Safety and Health Administration
ng	nanogram
NIOSH	National Institute for Occupational Safety and Health
NNK	4-(methyl-nitrosamino)-(3-pyridyl)-1-butanone
NRC	National Research Council
OSHA	Occupational Safety and Health Administration
P	probability
pCi	picocurie
PEL	permissible exposure limit
SC	suspected carcinogen
sec	second
SS	sidestream smoke (smoke generated by smoldering tobacco between puffs and smoke diffusing through the cigarette paper and escaping from the burning cone during puffing)
T	toxic
TP	tumor promoter
µg	microgram
µm	micrometer

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CURRENT INTELLIGENCE BULLETIN 54

ENVIRONMENTAL TOBACCO SMOKE IN THE WORKPLACE

Lung Cancer and Other Health Effects

INTRODUCTION

The Surgeon General has concluded that tobacco smoke is a carcinogen and an important risk factor for heart disease. The purpose of this bulletin is to disseminate information about the potential carcinogenicity of environmental tobacco smoke* (ETS) in the workplace. Evidence is now clear that the health risk from inhaling tobacco smoke is not limited to the smoker, but also includes those who inhale ETS. Recent epidemiologic studies of nonsmokers exposed to ETS have shown an increased relative risk for lung cancer compared with unexposed nonsmokers. In addition, recent evidence suggests that exposure of nonsmokers to ETS may be associated with an increased risk of heart disease. This bulletin describes the results and implications of these studies.

The conclusions and recommendations in this Current Intelligence Bulletin (CIB) are based on the following:

- Reports of the Surgeon General on the health effects of tobacco smoke
- Comparison of the chemical composition of ETS with that of mainstream smoke† (MS)
- Results from recent epidemiologic studies of nonsmokers exposed to ETS

Methods for controlling involuntary exposures to ETS in the workplace are also discussed.

REPORTS OF THE SURGEON GENERAL ON THE HEALTH EFFECTS OF TOBACCO SMOKE

In 1964, the Surgeon General issued the first report on smoking and health, which concluded that cigarette smoke causes cancer [DHEW 1964]:

> Cigarette smoking is causally related to lung cancer in men; the magnitude of the effect of cigarette smoking far outweighs all other factors. The data for women, though less extensive, point in the same direction. . . . The risk of developing lung cancer increases with duration of smoking and the number of cigarettes smoked per day, and is diminished by discontinuing smoking.

Since 1964, evidence has continued to support the causal relationship between exposure to cigarette smoke and lung cancer, demonstrating that risk increases with amount and duration of smoking. Subsequent research has increased our knowledge about the toxicity and carcinogenicity of tobacco smoke and the risks of exposure. Additional support for the Surgeon General's conclusion has

*Tobacco smoke in the ambient atmosphere composed of sidestream smoke and exhaled mainstream smoke. See page viii for a complete list of definitions and abbreviations.
†Smoke drawn through the tobacco and into the smoker's mouth.

come from (1) animal studies that demonstrated the carcinogenicity of tobacco smoke condensate, and (2) analytical studies demonstrating that tobacco smoke contains carcinogens [DHEW 1972; DHHS 1982]. Cigarette smoking is the major cause of lung cancer (87% of lung cancer deaths) and is estimated to account for 30% of all cancer deaths [DHHS 1989].

The 1964 Surgeon General's report also pointed out that male cigarette smokers have higher death rates from heart disease than nonsmokers. Subsequent reports have concluded that cigarette smoking is a major cause of heart disease and that smoking is a major independent risk factor for heart attack [DHEW 1968; DHHS 1983].

On July 1, 1965, Congress approved the Federal Cigarette Labeling and Advertising Act of 1965 (Public Law 89-92). This law, which became effective on January 1, 1966, was the first of a continuing series of Federal statutes enacting warning labels to inform the public about the health hazards of smoking and, subsequently, the use of other tobacco products. Presently, the Comprehensive Smoking Education Act (Public Law 98-474) [Title 15, § 1331 of the U.S. Code] requires cigarette companies to rotate four health warnings on all cigarette packages and in advertisements.

COMPARISON OF THE CHEMICAL COMPOSITION OF ETS AND MS

ETS contains many of the toxic agents and carcinogens that are present in MS, but in diluted form [DHHS 1986]. The major source of ETS is sidestream smoke* (SS), which contains higher amounts of some toxic and carcinogenic agents than MS when it is obtained in its undiluted form under laboratory conditions [DHHS 1989]. For example, the release of volatile N-nitrosamines and aromatic amines is higher in SS than in MS.

*Smoke generated by (1) smoldering tobacco between puffs, and (2) smoke diffusing through the cigarette paper and escaping from the burning cone during puffing.

A major reason that undiluted SS and MS have different concentrations of toxic and carcinogenic agents is that peak temperatures in the burning cone of a cigarette reach 800° to 900°C during puffing, but only 600°C between puffs, resulting in less complete combustion of tobacco during generation of SS. In addition, most of the burning cone is oxygen deficient during smoldering and produces a strongly reducing environment [NRC 1986]. Table 1 lists 26 toxic and carcinogenic agents identified in SS and MS.

ETS is diluted in the air before it is inhaled and thus is less concentrated than MS. However, active inhalation of MS is limited to the time it takes to smoke each cigarette, whereas exposure to ETS is constant over the period spent in the ETS-polluted environment. This fact is reflected in measurements of nicotine uptake by smokers and ETS-exposed nonsmokers [DHHS 1989].

POTENTIAL FOR OCCUPATIONAL EXPOSURE

Approximately 29% of the U.S. adult population smokes cigarettes, and exposure to ETS is common [DHHS 1989]. Many people who report no exposure to ETS have low concentrations of cotinine (a metabolite of nicotine) in their urine, indicating exposure. The average concentration of cotinine in the urine of nonsmokers has been reported to be approximately 8 ng/ml compared with approximately 1,200 ng/ml in smokers [Cummings et al. 1990]. The National Research Council (NRC) estimated that nonsmokers exposed to ETS averaged 25 ng of urinary cotinine/ml, and active smokers averaged 1,825 ng/ml [NRC 1986]. Husgafvel-Pursiainen et al. [1987] found that nonsmoking restaurant workers had an average urinary cotinine concentration of 56 ng/ml, and nonsmokers not exposed to ETS had an average concentration of 8.3 ng/ml. Other investigators have shown that nonsmokers living with smokers have approximately two to three times the amount of urinary cotinine as

Table 1.—Toxic and carcinogenic agents in undiluted cigarette SS*,†

Compound	Type of toxicity	Amount in SS (per cigarette)	Ratio of SS/MS
Vapor phase:			
Carbon monoxide	T	26.8–61 mg	2.5–14.9
Carbonyl sulfide	T	2–3 mg	0.03–0.13
Benzene	C	400–500 µg	8–10
Formaldehyde	C	1,500 µg	50
3-Vinylpyridine	SC	300–450 µg	24–34
Hydrogen cyanide	T	14–110 µg	0.06–0.4
Hydrazine	C	90 ng	3
Nitrogen oxides (NO_x)	T	500–2,000 µg	3.7–12.8
N-nitrosodimethylamine	C	200–1,040 ng	20–130
N-nitrosopyrrolidine	C	30–390 ng	6–120
Particulate phase:			
Tar	C	14–30 mg	1.1–15.7
Nicotine	T	2.1–46 mg	1.3–21
Phenol	TP	70–250 µg	1.3–3.0
Catechol	CoC	58–290 µg	0.67–12.8
o-Toluidine	C	3 µg	18.7
2-Naphthylamine	C	70 ng	39
4-Aminobiphenyl	C	140 ng	31
Benz(a)anthracene	C	40–200 ng	2–4
Benzo(a)pyrene	C	40–70 ng	2.5–20
Quinoline	C	15–20 µg	8–11
N'-nitrosonornicotine	C	0.15–1.7 µg	0.5–5.0
NNK	C	0.2–1.4 µg	1.0–22
N-nitrosodiethanolamine	C	43 ng	1.2
Cadmium	C	0.72 µg	7.2
Nickel	C	0.2–2.5 µg	13–30
Polonium-210	C	0.5–1.6 pCi	1.06–3.7

*Sources: DHHS [1989]; Hoffmann and Hecht [1989].

†Abbreviations: C, carcinogenic; CoC, cocarcinogenic; MS, mainstream smoke; SC, suspected carcinogen; SS, sidestream smoke; T, toxic; TP, tumor promoter; NNK, 4-(methyl-nitrosamino)-(3-pyridyl)-1-butanone.

nonsmokers living with nonsmokers [Haley et al. 1989].

Exposures to ETS were measured by respirable suspended particulates (≤2.5 μm) and averaged 242 $\mu g/m^3$ in public access buildings [First 1984; NRC 1986; Repace and Lowrey 1980, 1982]. Studies reviewed by Repace and Lowrey [1990] suggested a 62% probability of exposure to ETS for a nonsmoker in the workplace. However, the relative contribution of work versus home environments in ETS exposure has not been well quantified. In addition, social settings outside the workplace or the home (e.g., restaurants and bowling alleys) may contribute significantly to ETS exposure.

On the basis of urinary cotinine concentrations, the NRC [1986] concluded that nonsmokers exposed to ETS absorb the equivalent of 0.1 to 1.0 cigarette per day. On the basis of 1985 data, NIOSH estimates that each cigarette smoker in the United States smokes an average of about 21 cigarettes per day [NCHS 1988]. Blood and urine samples analyzed for vapor phase nicotine indicate that nonsmokers exposed to ETS absorb about 1% of the tobacco combustion products absorbed by active smokers [NRC 1986; DHHS 1986].

EPIDEMIOLOGIC STUDIES OF NONSMOKERS EXPOSED TO ETS

Lung Cancer

Surgeon General

The Surgeon General first addressed the possible health effects of involuntary smoking in 1972 [DHEW 1972]. Evidence associating adverse health effects with ETS exposure continued to be reported, and in 1986, the Surgeon General's report entitled *The Health Consequences of Involuntary Smoking* focused entirely on this subject [DHHS 1986]. This report cited 3 cohort studies and 10 case-control studies that together documented an approximately 1.3-fold increase in the risk of lung cancer among nonsmoking women, usually wives exposed to their husbands' ETS. The 1986 Surgeon General's report concluded that involuntary smoking is a cause of disease (including lung cancer) in healthy nonsmokers; however, more data on the dose and distribution of ETS exposure in the population are needed to accurately estimate the magnitude of risk in the U.S. population [DHHS 1986].

NRC [1986]

The NRC also reviewed the important epidemiologic data available in 1986 (3 cohort studies and 10 case-control studies) on the adverse health effects of ETS exposure. These studies were the same ones reviewed by the Surgeon General, except for one case-control study. The statistical power of a single study is often small, but it can be improved by analyzing all the data simultaneously across all studies (meta-analysis). Using this approach, the NRC estimated that the relative risk for nonsmokers married to smokers was 1.25 compared with nonsmokers married to nonsmokers, taking into account the possible misclassification of smokers as nonsmokers. Using data from urinary cotinine tests to estimate the extent of ETS exposure, the NRC also estimated that the relative risk of lung cancer for nonsmokers exposed to ETS at home, at work, or elsewhere was 1.42 compared with unexposed nonsmokers, with a plausible range of 1.24 to 1.61. The NRC concluded that "the weight of the evidence derived from epidemiologic studies shows an association between ETS exposure of nonsmokers and lung cancer that, taken as a whole, is unlikely to be due to chance or systematic bias."

Wald et al. [1986]

In another quantitative meta-analysis covering the same studies reviewed by the NRC, Wald et al. [1986] reached similar conclusions. Their analysis showed a 30% greater risk of lung cancer (relative risk 1.30) for nonsmokers living with smokers relative to nonsmokers living with

nonsmokers. The authors concluded that this result was unlikely to have occurred by chance and that it underestimated the true risk associated with exposure to ETS because nonsmokers living with nonsmokers are exposed to ETS in other settings (e.g., at work). The relative risk is thus based on a reference group that is partially exposed.

Blot and Fraumeni [1986]

Blot and Fraumeni [1986] analyzed the same studies reviewed by the Surgeon General and the NRC except for one case-control study. Combining data across the reviewed studies, these investigators concluded that the overall relative risk for nonsmoking women married to husbands who smoked was 1.3 (95% confidence interval [CI], 1.1-1.5) compared with nonsmoking women married to nonsmokers. These authors also concluded that the relative risk for nonsmokers exposed to heavy smoking was 1.7 (95% CI, 1.4-2.1). They found that the epidemiologic studies strongly suggested an increased risk that was biologically plausible, but that limitations in assessing ETS exposure had introduced uncertainty.

Eight additional studies of lung cancer and ETS exposure among those who never smoked have been published since the reviews by the Surgeon General [DHHS 1986; NRC 1986; Wald et al. 1986; Blot and Fraumeni 1986] (Table 2).

Hole et al. [1989]

Hole et al. [1989] updated an earlier cohort study [Gillis et al. 1984] of 3,960 men and 4,037 women in Scotland. These men and women had lived in the same households and had been followed for an average of 11.5 years. They were aged 45-64 at the time of the original interviews, which took place from 1972 to 1976. The unexposed group was defined as persons who never smoked and lived with nonsmokers at the time of interview. The exposed group was composed of persons who never smoked and lived with smokers. A third group was composed of persons who had smoked during some period in the past. In the followup study [Hole et al. 1989], only 2 deaths from lung cancer occurred in the unexposed group and 7 in the exposed group, compared with 147 deaths from lung cancer among smokers. The adjusted relative risk for the exposed group was 2.41 (95% CI, 0.45-12.83), compared with a relative risk of 10.64 for persons who had smoked in the past. The risk for smokers was the same whether or not they were exposed to ETS.

Brownson et al. [1987]

Brownson et al. [1987] conducted a case-control study of persons diagnosed with adenocarcinoma of the lung. A subset of 19 nonsmoking women was identified from this group and was compared with 47 controls. Exposure to ETS was classified as less than 4 hr/day or more than 4 hr/day (there was no specification of when this exposure occurred). The odds ratio for those exposed more than 4 hr/day was 1.68 (95% CI, 0.39-2.97) after adjustment for confounders, whereas the odds ratio for those exposed fewer than 4 hr/day was 1.00. The study does not state whether the nonsmoking females had ever smoked, but the implication is that they had never smoked. Because many of the subjects were deceased, smoking status was often determined from interviews with next of kin. The study lacked sufficient sample size to draw substantive conclusions, but it did suggest an increased risk for adenocarcinoma among nonsmokers exposed to ETS.

Humble et al. [1987]

Humble et al. [1987] evaluated the risk of lung cancer in a case-control study of 28 lung cancer patients who never smoked and lived with a smoking spouse compared with a control group of 54 persons who never smoked and lived with a nonsmoking spouse. Surrogate responses about smoking habits were used for 19 of the 28 cases, with most of these data provided by the spouse.

Table 2.—Recent studies of lung cancer among ETS-exposed persons who never smoked

Study	Design	Exposure definition	Relative risk*	Comment
Update of Gillis et al. [1984] by Hole et al. [1989]	12-yr followup, 3,960 men and 4,037 women aged 45–64 in 1972–76	Living with smoker or ex-smoker at the time of the survey	2.41 (CI, 0.45–12.83; 7 observed)	Adjusted for age, sex, and social class
Brownson et al. [1987]	19 cases, 47 controls	Exposure for ≥4 hr /day	1.68 (CI, 0.39–2.97)	Adjusted for age, income, and occupation
Humble et al. [1987]	28 cases, 54 controls	Lived with a spouse who smoked	For cigarette ETS exposure: 2.2 (90% CI, 1.0–4.9) For any type of ETS exposure: 2.6 (90% CI, 1.2–5.6)	Adjusted for ethnicity and age
Gao et al. [1987]	246 cases, 375 controls	Lived with a smoker	<20 yr, 1.0; 20–29 yr, 1.1 (CI, 0.7–1.8); 30–39 yr, 1.3 (CI, 0.8–2.1); ≥40 yr, 1.7 (CI, 1.0–2.9)	Adjusted for age and education
Lam et al. [1987]	199 cases, 335 controls	Lived with a spouse who smoked	1.65 (CI, 1.16–2.35)	Matched for age and neighborhood
Janerich et al. [1990]	191 cases, 191 controls	≥25 smoker-yr of exposure during childhood	2.07 (CI, 1.16–3.68)	Matched for age, sex, and county of residence; relative risk for spousal smoking = 0.9
Shimizu et al. [1988]	90 cases, 163 controls	Lived with mother who smoked Lived with father-in-law who smoked	4.0 ($P<0.05$) 3.2 ($P<0.05$)	Matched for age, sex, and hospital; relative risk for spousal smoking = 1.1
Geng et al. [1988]	54 cases, 93 controls	Lived with spouse who smoked	2.16 (CI, 1.03–4.53)	Matched for race, age, sex, and marital status; positive dose response; methodologic details not presented

*Confidence interval is 95% unless otherwise indicated.

The adjusted odds ratio for nonsmokers living with a spouse who smoked cigarettes was 2.2 (90% CI, 1.0-4.9), and it was 2.6 (90% CI, 1.2-5.6) for nonsmokers exposed to any type of ETS (including pipes and cigars). There was no trend of increasing risk with increased duration of exposure or increased amount regularly smoked by the spouse. In addition, marriage to a smoker did not increase the risk for persons who had ever smoked. This study contained no data on exposure to ETS outside the home or from other persons (nonspouses) living in the home.

Gao et al. [1987]

Gao et al. [1987] studied 672 female lung cancer patients and 735 population-based controls in Shanghai, China. Patients had been diagnosed as having lung cancer between 1984 and 1986, and both patients and controls were all interviewed directly. The odds ratio (adjusted for age and education) was 0.9 (95% CI, 0.6-1.4) for persons who had ever lived with a smoker during adulthood, and 1.1 (95% CI, 0.7-1.7) for those who had ever lived with a smoker during childhood. However, for 246 married patients and 375 married controls, risk increased with increasing years of marriage to a spouse who smoked, reaching 1.7 (95% CI, 1.0-2.9) among nonsmokers who lived with a smoker for more than 40 years.

Lam et al. [1987]

Lam et al. [1987] studied 445 female lung cancer patients in Hong Kong matched by age with 445 female controls from the same neighborhood. Analyses (unmatched) for exposure to ETS included 199 married patients and 335 married controls who never smoked. A small number of persons who were not married and never smoked (5% to 6% of the patients and controls) were also included and treated as unexposed to ETS. Wives exposed to ETS from a husband who smoked had an odds ratio of 1.65 (95% CI, 1.16-2.35), with the predominant type of cancer being adenocarcinoma (odds ratio 2.12). These odds ratios were not adjusted for any confounders.

Janerich et al. [1990]

Janerich et al. [1990] conducted a population-based case-control study of 191 persons who never smoked and were diagnosed with lung cancer from 1982 to 1984; this group was compared with individually matched controls who never smoked. Controls were matched by age, sex, and county of residence. Surrogate interviews were necessary for 62 cases and thus were also conducted for their matched controls. The number of years of exposure to ETS in the home was calculated by summing the number of smokers in the home per year across all years of life. The number of years exposed to ETS outside the home was also estimated, although details were not given in the report. Childhood and adolescent exposures in the home, adult exposures in the home, and adult exposures outside the home all contributed about equally to total lifetime exposure to ETS. For spouses, ETS exposure was also calculated by multiplying the number of packs smoked per day by the number of years the spouse smoked. The major finding of the study was a trend of increasing lung cancer risk with increasing years of exposure during childhood. Household exposure to ETS for 25 or more years that included childhood doubled the risk of lung cancer (odds ratio, 2.07; 95% CI, 1.16-3.68). No consistent increase in cancer risk occurred with increasing years of adulthood exposure to all sources of ETS, but persons exposed for the greatest number of years showed the highest risk (odds ratio, 1.11; 95% CI, 0.56-2.20). Separate analyses of exposure to ETS from spousal smoking found no excess risk of lung cancer for individuals married to smokers, and no trend of increased risk with increased years of exposure or with increased amount smoked by the spouse. Odds ratios in this study were not adjusted for any confounders, and odds ratios for exposure to ETS from spousal smoking do not appear to have been adjusted for childhood ETS exposures.

Shimizu et al. [1988]

Shimizu et al. [1988] published a case-control study of 90 female lung cancer patients who never smoked. Each patient was matched by age, hospital, and admission date to 2 controls of the same sex who did not have lung cancer (for 17 patients, only 1 control was used). Information was gathered about occupation, exposure to ETS, diet, and cooking fuels. The risk of lung cancer was significantly elevated for women who never smoked and lived in a home where the mother smoked (relative risk, 4.0; $P<0.05$) or the paternal grandfather smoked (relative risk, 3.2; $P<0.05$). Exposure to the ETS of other household members (including the husband, the father, and the children) was not associated with increased risk. The relative risk for exposure to ETS at work was 1.2. The authors found no increasing trend in risk with the number of cigarettes smoked by the mother or the paternal grandfather. However, the authors indicated that the subjects had trouble recalling the amount of smoking to which they had been exposed. The authors also pointed out that in Japan, children spend considerable time with their mother in the home, and less with the father; it is also common for the retired father of the husband to live in his son's home.

Geng et al. [1988]

Geng et al. [1988] studied 54 nonsmoking (undefined) women with lung cancer in Tianjin, China. These patients were matched with controls by race, age, sex, and marital status. The authors reported a significantly elevated odds ratio for the patients exposed to ETS from spouses (odds ratio, 2.16; 95% CI, 1.03-4.53), but it is not clear whether these women included former smokers. Their risk increased with the amount and duration (years) of the husband's smoking.

Summary of Results from Lung Cancer Studies

The studies published since 1986 have concentrated on ETS exposure through spousal smoking during adulthood [Hole et al. 1989; Brownson et al. 1987; Humble et al. 1987; Gao et al. 1987; Lam et al. 1987; Janerich et al. 1990; Shimizu et al. 1988; Geng et al. 1988], although some studies include data on childhood exposures [Janerich et al. 1990; Shimizu et al. 1988; Gao et al. 1987]. The data from these more recent studies do not individually demonstrate a clear causal relationship between ETS exposure and lung cancer, but their consistent finding of a relative risk greater than 1.0 for nonsmokers exposed to ETS provides evidence of a positive association. These data are consistent with and reinforce the reviews by NRC [1986], DHHS [1986], Blot and Fraumeni [1986], and Wald et al. [1986], all of which concluded that ETS exposure is associated with an increased risk of lung cancer for nonsmokers.

The most important limitation observed in all studies reporting lung cancer risks among persons who never smoked is the lack of quantitative ETS exposure data. Most studies have defined exposure to ETS for nonsmokers on the basis of living with or being married to a smoker. All of the case-control studies ascertained the potential for exposure by interviewing subjects and controls (or surrogates) without any other independent assessment of ETS exposure. Questionnaires often failed to include specific questions about all sources of ETS. Most studies included limited or no information about the risk of lung cancer from ETS exposure in the workplace.

The potential exists for a positive bias, particularly in the case-control studies, where ex-smokers with lung cancer might have been misclassified as never having smoked. The misclassified ex-smokers are more likely to be married to smokers and to develop lung cancer than those who never smoked. However, the NRC [1986] estimated that the effect of such misclassification would have been relatively slight and could not entirely account for the increased risk of lung cancer following exposure to ETS. This conclusion by the NRC is based on calculations that assume a degree of misclassification (based on nonsmoker urinary cotinine data)

and its likely effect on the observed lung cancer risk. In addition, the risk of lung cancer in these studies is based on a reference group of nonsmokers living with nonsmokers who are exposed to ETS in other settings. This background exposure results in underestimation of the true risk.

Several risk assessments have been performed for persons who never smoked and were exposed to ETS. The NRC [1986] estimated that of the 7,000 lung cancer deaths reported in 1985 among U.S. women who never smoked, 2,010 (29%) were attributable to ETS. The corresponding number for men was 820 (16%) of the 5,200 lung cancer deaths among U.S. males who never smoked. Wells [1988] estimated that approximately 3,000 of the lung cancer deaths reported in 1985 among persons who never smoked occurred as a result of ETS exposure. Repace and Lowrey [1990] estimated that approximately 5,000 of the lung cancer deaths reported in 1988 among persons who never smoked and ex-smokers occurred as a result of ETS exposure. These risk assessments used a 1.3 to 1.5 relative risk of lung cancer for nonsmokers exposed to ETS at home or elsewhere compared with unexposed persons. The number of deaths attributable to ETS exposure was derived using standard formulas for attributable risks [Kleinbaum et al. 1983] with the estimated number of annual lung cancer deaths in the United States for persons who have never smoked.

Heart Disease

Surgeon General

The Surgeon General [DHHS 1986] reviewed four epidemiologic studies of cardiovascular disease in persons exposed to ETS [Lee et al. 1986; Hirayama 1984; Gillis et al. 1984; Garland et al. 1985] (Table 3). He concluded that "further studies on the relationship between involuntary smoking and cardiovascular disease are needed in order to determine whether involuntary smoking increases the risk of cardiovascular disease. More detailed characterizations of exposure to ETS and specific types of cardiovascular disease associated with this exposure are needed before an effect of involuntary smoking on the etiology of cardiovascular disease can be established."

NRC [1986]

The NRC [1986] reviewed four epidemiologic studies that evaluated the association between heart disease and ETS exposure [Garland et al. 1985; Gillis et al. 1984; Hirayama 1984; Svendsen et al. 1985, 1987*] (Table 3). The same NRC review examined studies of the effects of ETS exposure on heart rate and blood pressure among healthy individuals. No statistically significant increases were found in heart disease or effects on heart rate and blood pressure. The NRC report concluded that "with respect to chronic cardiovascular morbidity and mortality, although biologically plausible, there is no evidence of statistically significant effects due to ETS exposure, apart from the study by Hirayama [see Table 3] in Japan."

Other Epidemiologic and Experimental Studies

Since publication of the reports by the NRC and the Surgeon General, researchers have published two epidemiologic studies of heart disease and ETS exposure [Humble et al. 1990; Helsing et al. 1988], and one update [Hole et al. 1989] of an earlier cohort study [Gillis et al. 1984] (Table 3). Experimental studies of the effects of ETS on the heart have also been published [Davis et al. 1989; Allred et al. 1989; Sheps et al. 1990].

The studies reported by Hole et al. [1989], Helsing et al. [1988], and Humble et al. [1990] associated ETS exposure with an increase of heart disease among persons who never smoked. The studies by Hole et al. [1989] and Helsing et al. [1988] are both large follow-up cohort studies that used direct interviews of men and women who lived in the same household. Study of these cohorts demonstrated

*Svendsen et al. [1985] is the abstract of the full report published in 1987 [Svendsen et al. 1987].

Table 3.—Recent studies of heart disease among ETS-exposed persons who never smoked

Study	Design	Exposure definition	Relative risk*	Comment
Update of Gillis et al. [1984] by Hole et al. [1989]†	12-yr followup, 3,960 men and 4,037 women aged 45-64 in 1972-76	Living with smoker or ex-smoker in early 1970s	2.01 (CI, 1.21-3.35; 485 observed)	Adjusted for cardiovascular risk factors,§ positive dose response
Humble et al. [1990]	20-yr followup, 513 women aged 40+	Living with smoker in 1960	1.59 (CI, 0.99-2.57)	Adjusted for cardiovascular risk factors, dose response in some strata
Helsing et al. [1988]	12-yr followup, 4,162 men and 14,873 women, aged 25+ in 1963	Living with smoker or ex-smoker in 1963	Men: 1.31 (CI, 1.1-1.6; 492 observed) Women: 1.24 (CI, 1.1-1.4; 1,539 observed)	Adjusted for education, positive dose response among women only
Svendsen et al. [1985, 1987]**	7-yr average followup, 1,245 men aged 35-57 in 1973-82	Married to smoker or ex-smoker	1.61 (CI, 0.96-2.71; 90 observed)	Adjusted for cardiovascular risk factors, positive dose response
Garland et al. [1985]	10-yr followup, 695 women aged 50-79 in 1972-74	Married to smoker or ex-smoker	2.9 (estimate; 19 observed)	Adjusted for age
Hirayama [1984]	16-yr followup, 91,540 women aged 40+	Married to smoker or ex-smoker	Low exposure: 1.10 (90% CI, 0.91-1.33) High exposure: 1.31 (90% CI, 1.06-1.93; 494 observed)	Significant dose response
Lee et al. [1986]	48 cases, 182 controls	Married to smoker or ex-smoker	Men: 1.24 Women: 0.93	No apparent dose response

*Confidence interval is 95% unless otherwise indicated.
†Hole et al. [1989] provide updated results of the same population studied by Gillis et al. [1984].
§Serum cholesterol, blood pressure, and body mass index.
**Svendsen et al. [1987] is the full report of the abstract published by Svendsen et al. [1985].

an excess of heart disease in persons who lived with smokers and never smoked compared with persons who lived with nonsmokers and never smoked. Furthermore, Hole et al. [1989] and Humble et al. [1990] show an increasing risk for heart disease mortality with increasing exposure to ETS at home. Helsing et al. [1988] found a similar trend in women but not men.

Experimental studies support the hypothesis that ETS exposure has deleterious effects on platelets and the endothelium [Davis et al. 1989] and can decrease the time to onset of angina pectoris in patients with coronary artery disease [Allred et al. 1989]. Allred et al. [1989] reported that the time to angina decreased in heart disease patients who exercised after exposure to airborne carbon monoxide (CO) at concentrations producing 2% and 3.9% carboxyhemoglobin (COHb) in the blood. Sheps et al. [1990] observed that arrhythmias in heart disease patients increased when they exercised after exposure to airborne CO at concentrations producing 6% COHb in the blood. Persons exposed to ETS in unventilated areas have been shown to have COHb concentrations of 2% to 3% [NRC 1986].

Wells [1988] estimated that among nonsmokers in the United States, 32,000 deaths from heart disease each year were attributable to ETS exposure. Wells used estimated rates for death from heart disease among nonsmokers and a relative risk of 1.30 for ETS-exposed nonsmokers compared with unexposed nonsmokers.

Glantz and Parmley [1991] recently reviewed the epidemiologic literature on exposure to ETS and heart disease (including the studies in Table 3) from 1984 to the present. These researchers estimated a 30% increase (relative risk, 1.30; 95% CI, 1.2–1.4) in the risk of death from ischemic heart disease or myocardial infarction in nonsmoking individuals exposed to ETS at home. Glantz and Parmley also noted that several of these studies found a dose-response relation between the amount of smoking by the spouse and the risk of heart disease in the nonsmoking spouse; they concluded that exposure to ETS can cause heart disease.

Summary of Results from Heart Disease Studies

The principal limitation found in the lung cancer studies also applies to the studies of heart disease in persons exposed to ETS—that is, the indirect method of assessing exposure to ETS (usually defined as spousal smoking). The second limitation of these heart disease studies is the difficulty in controlling for all known cardiovascular risk factors (e.g., blood pressure, serum cholesterol, and body mass index). In addition, the risk of heart disease for ETS-exposed persons who never smoked seems large compared with the risk of heart disease for smokers. Unlike lung cancer mortality, where the relative risk for smokers compared with nonsmokers is 22.4 for men and 11.9 for women, the relative risk of heart disease mortality for smokers compared with nonsmokers is 1.9 for men and 1.8 for women [DHHS 1989]. Note that the nonsmoking comparison group for these risk estimates includes those exposed to ETS.

Other Adverse Health Effects

Several additional adverse health effects have been associated with ETS exposure, including cervical cancer [Slattery et al. 1989], ischemic stroke [Donnan et al. 1989], spontaneous abortion [Ahlborg 1990], and low birthweight [NRC 1986]. However, evidence is insufficient to draw conclusions about the relationship of ETS exposure to these health effects.

CONCLUSIONS

In 1964 the Surgeon General concluded that cigarette smoke causes lung cancer. Since that time, additional research on the toxicity and carcinogenicity of tobacco smoke has demonstrated that the health risks from inhaling tobacco smoke are not limited to smokers, but also include those

who inhale ETS. ETS contains many of the toxic agents and carcinogens found in MS, but in diluted form. Recent epidemiologic studies support and reinforce the conclusions of the reviews by the Surgeon General and the NRC demonstrating that exposure to ETS can cause lung cancer. These reviews estimated the relative risk for lung cancer to be approximately 1.3 for nonsmokers living with smokers compared with nonsmokers living with nonsmokers. In addition, recent evidence also suggests a possible association between exposure to ETS and an increased risk for heart disease in nonsmokers. The recent epidemiologic studies (including those associating ETS with other adverse health effects) point to a pattern of health effects that is similar for both smokers and nonsmokers exposed to ETS.

NIOSH recognizes that these recent epidemiologic studies have several shortcomings: lack of objective measures for characterizing and quantifying exposures, failure to adjust for all confounding variables, potential misclassification of exsmokers as nonsmokers, unavailability of comparison groups that have not been exposed to ETS, and low statistical power. Nonetheless, NIOSH has determined that the collective weight of evidence (i.e., that from the Surgeon General's reports, the similarities in composition of MS and ETS, and the recent epidemiologic studies) is sufficient to conclude that ETS poses an increased risk of lung cancer and possibly heart disease to occupationally exposed workers. The epidemiologic data are not sufficient to draw conclusions about other health effects such as cervical cancer, ischemic stroke, spontaneous abortion, and low birthweight.

RECOMMENDATIONS

Several systems exist for classifying a substance as a carcinogen. Such classification systems have been developed by NTP [1989], IARC [1987], and OSHA [29 CFR 1990]. NIOSH considers the OSHA classification system (Identification, Classification, and Regulation of Potential Occupational Carcinogens [29 CFR 1990], also known as the OSHA carcinogen policy) the most appropriate for use in identifying occupational carcinogens.* The Surgeon General has concluded that cigarette smoke causes lung cancer as well as heart disease. Table 1 lists 21 known or suspected carcinogens, cocarcinogens, and tumor promoters identified as components of ETS and MS in analytical studies. Furthermore, a large body of evidence indicates that exposure to ETS has produced lung cancer in nonsmokers. NIOSH therefore considers ETS to be a potential occupational carcinogen in conformance with the OSHA carcinogen policy [29 CFR 1990].

The risk of developing cancer should be decreased by minimizing exposure to ETS. Employers should therefore assess conditions that may result in worker exposure to ETS and take steps to reduce exposures to the lowest feasible concentration.

METHODS FOR CONTROLLING INVOLUNTARY EXPOSURE TO ETS

Workers should not be involuntarily exposed to tobacco smoke. To prevent worker exposures to any hazardous substance, employers should first eliminate hazardous workplace emissions at their source. If elimination is not possible, emissions should be removed from the pathway between the source and the worker [NIOSH 1983]. Therefore, the best method for controlling worker exposure to ETS is to eliminate tobacco use from the workplace and to implement a smoking cessation program. Until tobacco use can be

*"'Potential occupational carcinogen' means any substance, or combination or mixture of substances, which causes an increased incidence of benign and/or malignant neoplasms, or a substantial decrease in the latency period between exposure and onset of neoplasms in humans or in one or more experimental mammalian species as the result of any oral, respiratory or dermal exposure, or any other exposure which results in the induction of tumors at a site other than the site of administration. This definition also includes any substance which is metabolized into one or more potential occupational carcinogens by mammals" [29 CFR 1990.103].

completely eliminated, employers should protect nonsmokers from ETS by isolating smokers. Methods for eliminating tobacco use from the workplace and isolating smokers are described here briefly.

Eliminating Tobacco Use from the Workplace

Worker exposure to ETS is most efficiently and completely controlled by simply eliminating tobacco use from the workplace. To facilitate elimination of tobacco use, employers should implement smoking cessation programs. The Association of Schools of Public Health (ASPH) has recommended the following strategy for smoking cessation [NIOSH 1986]. Specifically, management and labor should work together to develop appropriate nonsmoking policies that include some or all of the following:

- Prohibit smoking at the workplace and provide sufficient disincentives for those who do not comply
- Distribute information about health promotion and the harmful effects of smoking
- Offer smoking-cessation classes to all workers
- Establish incentives to encourage workers to stop smoking

Further information regarding workplace smoking policies and smoking cessation programs can be found in *No Smoking: A Decision Maker's Guide to Reducing Smoking at the Worksite* [American Cancer Society et al. 1985].

Isolating Smokers

The 1986 Surgeon General's report on involuntary smoking concluded that, "the simple separation of smokers and nonsmokers within the same airspace may reduce, but does not eliminate, the exposure of nonsmokers to ETS." In indoor workplaces where smoking is permitted, ETS can spread throughout the airspace of all workers. The most direct and effective method of eliminating ETS from the workplace is to prohibit smoking in the workplace. Until that is achieved, employers can designate separate, enclosed areas for smoking, with separate ventilation. Air from this area should be exhausted directly outside and not recirculated within the building or mixed with the general dilution ventilation for the building. Ventilation of the smoking area should meet general ventilation standards, and the smoking area should have slight negative pressure to ensure airflow into the area rather than back into the airspace of the workplace [ASHRAE 1989]. Guidance for designing local exhaust ventilation systems can be found in *Recommended Industrial Ventilation Guidelines* [Hagopian and Bastress 1976], *Industrial Ventilation—A Manual of Recommended Practice* [ACGIH 1986], and *Fundamentals Governing the Design and Operation of Local Exhaust Systems* [ANSI 1979].

Warning signs should be posted at the entrances to the workplace in both English and the predominant language of non-English-reading workers. These signs should state that smoking is prohibited or permitted only in designated smoking areas. If designated smoking areas are provided, they should be clearly identified by signs.

RESEARCH NEEDS

Research is needed to investigate the following issues:

- More accurate quantification of the increased risk of lung cancer associated with ETS exposure, including determination of other contributing factors (e.g., occupational exposures) that may accentuate the risk
- Determination of the concentration and distribution of ETS components in the workplace to help quantify the risk for the U.S. working population

- The association of ETS exposure with cancer other than lung cancer
- The relationship between ETS exposure and cardiovascular disease
- The relationship between ETS exposure and nonmalignant respiratory diseases such as asthma, bronchitis, and emphysema, and the effects of ETS on lung function and respiratory symptoms
- Possible mechanisms of ETS damage to the cardiovascular system, such as increased platelet aggregation, increased COHb leading to oxygen deprivation, or damage to the endothelium
- Effects of workplace smoking restrictions on the ETS exposure of nonsmokers and ETS-related health effects in nonsmokers

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APPENDIX
POSITIONS OF OTHER AGENCIES REGARDING ETS

The Occupational Safety and Health Administration (OSHA) and the Mine Safety and Health Administration (MSHA) have not established permissible exposure limits (PELs) for ETS in the workplace. OSHA is now preparing to address this issue as part of an indoor air quality standard.

The U.S. Environmental Protection Agency (EPA) states that ETS is a known cause of lung cancer and respiratory symptoms and that it has been linked to heart disease. EPA also recommends that exposure to ETS be minimized wherever possible by restricting smoking to separately ventilated areas directly exhausted to the outside, or by entirely eliminating smoking in buildings [EPA 1989].

The International Agency for Research on Cancer [IARC 1986] stated that epidemiologic studies have demonstrated an increased risk of lung cancer for nonsmoking spouses of smokers. Although researchers had substantial difficulty in determining exposure to ETS and other risk factors for the cancers studied, IARC concluded that "passive smoking gives rise to some risk of cancer." IARC also concluded that "there is sufficient evidence that tobacco smoke is carcinogenic to humans," and that "there is sufficient evidence that inhalation of tobacco smoke as well as topical application of tobacco smoke condensate cause cancer in experimental animals."

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United States Environmental Protection Agency | Research and Development (RD-689) | Air and Radiation (6203J)

January 1993

EPA

Respiratory Health Effects of Passive Smoking

Fact Sheet

Summary

The U.S. Environmental Protection Agency (EPA) has published a major assessment of the respiratory health risks of passive smoking (*Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders; EPA/600/6-90/006F*). The report concludes that exposure to environmental tobacco smoke (ETS) -- commonly known as secondhand smoke -- is responsible for approximately 3,000 lung cancer deaths each year in nonsmoking adults and impairs the respiratory health of hundreds of thousands of children.

Background

EPA studies of human exposure to air pollutants indicate that indoor levels of many pollutants often are significantly higher than outdoor levels. These levels of indoor air pollutants are of particular concern because it is estimated that most people spend approximately 90 percent of their time indoors.

In recent years, comparative risk studies performed by EPA and its Science Advisory Board have consistently ranked indoor air pollution among the top five environmental risks to public health. EPA, in close cooperation with other federal agencies and the private sector, has begun a concerted effort to better understand indoor air pollution and to reduce peoples' exposure to air pollutants in offices, homes, schools and other indoor environments where people live, work and play.

Tobacco smoking has long been recognized as a major cause of death and disease, responsible for an estimated 434,000 deaths per year in the United States. Tobacco use is known to cause lung cancer in humans, and is a major risk factor for heart disease.

In recent years, there has been concern that non-smokers may also be at risk for some of these health effects as a result of their exposure ("passive smoking") to the smoke exhaled by smokers and smoke given off by the burning end of cigarettes.

As part of its effort to address all types of indoor air pollution, in 1988, EPA's Indoor Air Division requested that EPA's Office of Research and Development (ORD) undertake an assessment of the respiratory health effects of passive smoking. The report was prepared by ORD's Office of Health and Environmental Assessment.

The document has been prepared under the authority of Title IV of Superfund (The Radon Gas and Indoor Air Quality Research Act of 1986), which directs EPA to conduct research and disseminate information on all aspects of indoor air quality.

Public and Scientific Reviews

A draft of this assessment was released for public review in June 1990. In December 1990, EPA's Science Advisory Board, a committee of independent scientists, conducted a review of the draft report and submitted its comments to the EPA Administrator in April 1991. In its comments, the SAB's Indoor Air Quality/Total Human Exposure Committee concurred with the primary findings of the report, but made a number of recommendations for strengthening it.

Incorporating these recommendations, the Agency again transmitted a new draft to the SAB in May of 1992 for a second review. Following a July 1992 meeting, the SAB panel endorsed the major conclusions of the report, including its unanimous endorsement of the classification of environmental tobacco smoke (ETS) as a Group A (known human) carcinogen.

EPA also received and reviewed more than 100 comments from the public, and integrated appropriate revisions into the final risk assessment.

Major Conclusions

Based on the weight of the available scientific evidence, EPA has concluded that the widespread exposure to environmental tobacco smoke in the U.S. presents a serious and substantial public health risk.

In adults:

- ETS is a human lung carcinogen, responsible for approximately 3,000 lung cancer deaths annually in U.S. nonsmokers. ETS has been classified as a Group A carcinogen under EPA's carcinogen assessment guidelines. This classification is reserved for those compounds or mixtures which have been shown to cause cancer in humans, based on studies in human populations.

In children:

- ETS exposure increases the risk of lower respiratory tract infections such as bronchitis and pneumonia. EPA estimates that between 150,000 and 300,000 of these cases annually in infants and young children up to 18 months of age are attributable to exposure to ETS. Of these, between 7,500 and 15,000 will result in hospitalization.

- ETS exposure increases the prevalence of fluid in the middle ear, a sign of chronic middle ear disease.

- ETS exposure in children irritates the upper respiratory tract and is associated with a small but significant reduction in lung function.

- ETS exposure increases the frequency of episodes and severity of symptoms in asthmatic children. The report estimates

that 200,000 to 1,000,000 asthmatic children have their condition worsened by exposure to environmental tobacco smoke.

• ETS exposure is a risk factor for new cases of asthma in children who have not previously displayed symptoms.

Scope of the Report

In 1986, the National Research Council (NRC) and the U.S. Surgeon General independently assessed the health effects of exposure to ETS. Both of these reports concluded that ETS can cause lung cancer in adult non-smokers and that children of parents who smoke have increased frequency of respiratory symptoms and lower respiratory tract infections. The EPA scientific assessment builds on these reports and is based on a thorough review of all of the studies in the available literature.

Since 1986, the number of studies which examine these issues in human populations has more than doubled, resulting in a larger database with which to conduct a comprehensive assessment of the potential effects which passive smoking may have on the respiratory health of adults as well as children.

Because only a very small number of studies on the possible association between exposure to secondhand smoke and heart disease and other cancers existed in the scientific literature at the time this assessment was first undertaken, EPA has not conducted an assessment of the possible association of heart disease and passive smoking. EPA is considering whether such an assessment should be undertaken in the future, but has no plans to do so at this time.

Scientific Approach

EPA reached its conclusions concerning the potential for ETS to act as a human carcinogen based on an analysis of all of the available data, including more than 30 epidemiologic (human) studies looking specifically at passive smoking as well as information on active or direct smoking. In addition, EPA considered animal data, biological measurements of human uptake of tobacco smoke components and other available data. The conclusions were based on what is commonly known as the total "weight-of-evidence" rather than on any one study or type of study.

The finding that ETS should be classified as a Group A carcinogen is based on the conclusive evidence of the dose-related lung carcinogenicity of mainstream smoke in active smokers and the similarities of mainstream and sidestream smoke given off by the burning end of the cigarette. The finding is bolstered by the statistically significant exposure-related increase in lung cancer in nonsmoking spouses of smokers which is found in an analysis of more than 30 epidemiology studies that examined the association between secondhand smoke and lung cancer.

The weight-of-evidence analysis for the noncancer respiratory effects in children is based primarily on a review of more than 100 studies, including 50 recent epidemiology studies of children whose parents smoke.

Beyond the Risk Assessment

Although EPA does not have any regulatory authority for controlling ETS, the

Agency expects this report to be of value to other health professionals and policymakers in taking appropriate steps to minimize peoples' exposure to tobacco smoke in indoor environments.

In cooperation with other government agencies, EPA will carry out an education and outreach program over the next two years to inform the public and policy makers on what to do to reduce the health risks of ETS as well as other indoor air pollutants.

For Further Information

A limited number of copies of the complete report can be obtained free of charge from:

Center for Environmental Research
Information (CERI)
U.S. EPA
26 W. Martin Luther King Drive
Cincinnati, OH 45268
Telephone: 513-569-7562
Fax: 513-569-7566

Ordering Number: EPA/600/6-90/006F

or

U.S. Environmental Protection Agency
Indoor Air Quality Information
Clearinghouse (IAQ INFO)
P.O. Box 37133
Washington D.C. 20013-7133
Telephone: 1-800-438-4318
Fax: 301-588-3408

A number of government agencies can provide additional information addressing the health risks of environmental tobacco smoke. These include:

Office on Smoking and Health/Centers for Disease Control
Center for Chronic Disease Prevention and Health Promotion
Mail Stop K-50, 4770 Buford Highway
Atlanta, GA 30341

National Cancer Institute
Building 31, Room 10A24
Bethesda, MD 20892
1-800-4-CANCER

The National Heart, Lung, and Blood Institute
Information Center
4733 Bethesda Avenue, Suite 530
Bethesda, MD 20814

National Institute for Occupational Safety and Health
4676 Columbia Parkway
Cincinnati, Ohio 45226-1998
1-800-35-NIOSH

HOUSE PUBLIC WORKS AND TRANSPORTATION COMMITTEE
SUBCOMMITTEE ON PUBLIC BUILDINGS AND GROUNDS

Statement of Gio Gori, Ph.D. on H.R. 881

Mister Chairman, Members of the Committee:

My name is Gio Gori. I am a consulting toxicologist with training in epidemiology and broad interests in smoking and health, cancer causation and risk assessment, and over 100 scientific publications to my credit. I am President of the International Society of Regulatory Toxicology and Pharmacology. In the 1960's and 1970's, I was Deputy Director for Cancer Cause and Prevention at the National Cancer Institute, where I received the Public Health Superior Service Award in 1977 for activities as Director of the Smoking and Health Program. My full curriculum vitae is appended to the written statements submitted at this hearing.

Because this bill appears to have been drafted in response to the report on environmental tobacco smoke (ETS) released by the Environmental Protection Agency (EPA) on January 7, 1993, The Tobacco Institute has asked me to explain my

concerns as a scientist about the report. My views are my own and are not necessarily those of The Tobacco Institute.

The EPA report is a glaring example of the misuse of science in support of preconceived policy aims. It is not unique, but it is possibly the most egregious example in a well-documented tradition of scientific abuse at EPA.

In 1991, an independent blue ribbon panel was convened by then-Administrator Reilly to investigate accusations of "junk science" at the Agency. The panel concluded that the Agency does not have a coherent science agenda and suffers from poor scientific credibility because it has not secured adequate external support from the scientific community at large. More to the point, the panel concluded that EPA's science often seems to have been adjusted to endorse policy, an especially relevant statement when considering the Agency's report on ETS.

EPA's own research director, Dr. Erich Bretthauer, recently lamented that the agency too often pays more attention to media headlines than to scientific evidence. And at the press briefing following the release of the ETS report, Dr. Bretthauer acknowledged that the EPA's $26 billion cost estimate for the Clean Air Act implementation may prevent fewer than 100 lung cancer cases a year, and perhaps none at all. This is a typical

example of overregulation by EPA without solid scientific support.

As far as ETS is concerned, the Agency's staff disclosed its predisposition with respect to ETS long before the report was even drafted. In 1989 EPA disseminated a so-called "fact sheet" asserting that ETS was a known cause of lung cancer and other diseases. The same claim was made in a 1990 draft policy guide aimed at banning smoking in workplaces. In its final report itself, the Agency had no hesitation in writing that its analysis was based "on the a priori hypothesis . . . that a positive association exists between exposure to ETS and lung cancer." (1, p. 5-2).

The agency and its Science Advisory Board conceded that ETS could be classified as a Group A carcinogen only if the Agency's guidelines for carcinogen assessment -- guidelines that were carefully developed according to firmly established scientific principles -- were disregarded. In a telling statement, the SAB panel reviewing the ETS risk assessment declared: "If the guidelines for Carcinogen Risk Assessment can be used to cast doubt on a finding that inhalation of tobacco smoke by humans causes an increased risk of lung cancer, the situation suggests a need to revise the guidelines." (2, p. 28).

A first requirement of EPA's guidelines -- and also an intuitively correct one -- is that the substance considered be positively identified. However, what substance is EPA dealing with? The report itself is forced mostly to speculate about ETS components. Only about twenty have been identified with some confidence as being found in ETS, although most could come from other sources as well. Other components cannot be measured because of extreme dilution. EPA solved the problem by arbitrarily declaring that ETS is equivalent to the smoke that smokers inhale.

Never mind that the agency's Science Advisory Board admonished EPA's staff that the alleged equivalency of ETS and cigarette smoke could not be scientifically sustained. Never mind that the EPA report itself discounts this similarity in its fine print. Never mind that exposures to ETS components are tens of thousands to a million fold less than for cigarette smokers, and far below the levels permitted for such components in workplaces by the Occupational Safety and Health Administration (3).

In fact, if EPA were correct with regard to ETS it would be necessary, for the sake of consistency, to designate as human carcinogens mixtures such as vehicle exhausts, wood and gas fires in homes, grill and barbecue fumes and others, simply because they bear similarities to coke oven emissions, which EPA

already classifies as known human carcinogens. In truth, this contrived identification of ETS with mainstream cigarette smoke was needed to bolster the weak and inconclusive results of ETS epidemiologic studies. Of 30 such studies from all over the world, only six reported significant associations between marriage to a smoker and lung cancer risk, and some actually reported decreased risks. The EPA based its risk assessment on 11 US studies, none of which was statistically significant overall.

To claim significance, however, EPA resorted to what the Agency staff acknowledged was "fancy statistical footwork." The gambit involved doubling the statistical "confidence interval" from the standard $\pm$ 5% point margin usually employed -- even in the first draft of the EPA report -- to a $\pm$ 10% point margin. Naturally, as if by magic the results became statistically significant. Yet, what any self-respecting scientist would perceive here is not confidence intervals, but rather a confidence game.

There is more. Two months before the EPA risk assessment was released, two new epidemiologic studies addressing ETS and the risk of lung cancer were published in the scientific literature. One of these, funded in part by the National Cancer Institute, is the largest case-control study published to date in the U.S. When the data from these two studies are added to EPA's

analysis, the result is not statistically significant based on a standard margin of error or even on EPA's less rigorous standard. Yet EPA refused to consider this latest and clearly relevant information. It also ignored that 13 out of 15 epidemiologic studies of ETS in workplaces failed to detect any elevation of lung cancer risk.

In epidemiology the 5% standard is universally prescribed as a minimum requirement. This is especially true for epidemiologic studies of potential low level risks for lung cancer, which are notoriously difficult to conduct and interpret. For instance, there is firm evidence that differences in diet, physical activity, disease experience, socioeconomic status, occupation, and other variables can influence the small reported association between marriage to a smoker and increased risk of lung cancer. One cannot look at ETS alone, as the agency has done.

To confirm EPA's unjustified conclusion with respect to ETS, consider only that the agency dismissed the epidemiologic evidence on electromagnetic fields, even though 25 of some 40 available studies were statistically significant and reported cancer risks substantially larger that those attributed to ETS.

Thus, EPA's claim that ETS causes some 3000 lung cancer deaths a year is a political conclusion shored up by an assumed

identity, unwarranted assumptions, selective use of data, artful statistical manipulations, and the contrived illusion of mathematical precision. On scientific grounds, a zero excess of lung cancer is just as tenable as -- if not more tenable than -- EPA's creations.

We may ask: why is the agency doing this? Its immediate interests involve expanding the support for an army of employees, and reinforcing the financial and political clout of its management and its many influential advocates. Since the agency itself was funded because of public concern, it must keep stoking the fires of fear. As former Administrator Reilly's expert panel noted in its critique of EPA science, "The legal process fosters the presentation of the extremes of scientific opinion." We cannot forget EPA's periodic, costly, but exaggerated reports about ethylene dibromide, Alar, dioxin, environmental asbestos, radon, and other toxic contaminants of food, air and water.

Even among the scientists who reviewed the EPA report there are those whose scientific writings reflect disagreement with the report's conclusions, even though they apparently feel the need to agree in public with an Agency that influences much of the research funding they obtain (4). EPA and its associates must believe that the end justifies whatever means in the crusade against tobacco. But who could feel safe if such an attitude

were applied generally? In the end, who will control the controllers?

A regulatory agency does not fulfill the public's expectations of due process if it operates under its own rules on the basis of unwarranted assumptions. In an open society such an agency should not be allowed to act arbitrarily. At the very least, it should be required to obey generally accepted norms of scientific evidence and conduct: norms that are independently justified by their transparent and rational fairness, and their timeless ethical values.

Tobacco and smoking may indeed be legitimate public health issues, but the ETS report does not rest on firm scientific ground. The EPA report on ETS flies in the face of the standards of conduct that all scientists are required to observe.

Mr. Chairman, that concludes my statement. I will be happy to answer your questions.

References

1. United States Environmental Protection Agency. Respiratory health effects of passive smoking: Lung cancer and other disorders. Washington, DC, December 1992.

2. United States Environmental Protection Agency. Review of the Office of Research and Development's draft report: "Health effects of passive smoking: assessment of lung cancer in adults and respiratory diseases in children". April 1991.

3. Gori, G.B., Mantel, N., Mainstream and environmental tobacco smoke. Regul Toxicol Pharmacol 14:88-105, 1991.

4. United States Environmental Protection Agency. Science Advisory Board. IAQTHEC Committee. Environmental Tobacco Smoke Review. Days Inn Hotel, Arlington, Virginia December 4, 1990.

TESTIMONY OF

MARK W. HURWITZ, PH.D., CAE, REPRESENTING

THE BUILDING OWNERS AND MANAGERS ASSOCIATION (BOMA)

INTERNATIONAL

BEFORE THE

SUBCOMMITTEE ON PUBLIC BUILDINGS AND GROUNDS

HOUSE PUBLIC WORKS AND TRANSPORTATION COMMITTEE

HEARING ON H.R. 881

THE "BAN ON SMOKING IN FEDERAL BUILDINGS ACT"

MARCH 11, 1993

The Building Owners and Managers Association International
1201 New York Avenue, NW, Suite 300
Washington, DC 20005
202-408-2662

Good morning, Mr. Chairman and Members of the Subcommittee. My name is Mark Hurwitz, and I am Executive Vice President of the Building Owners and Managers Association International (BOMA). BOMA is the nation's largest and oldest trade association exclusively representing the office building industry. Our membership owns or manages over 5 billion square feet of commercial office space in the United States.

Thank you for the opportunity to present testimony today. We commend your Subcommittee for addressing this important issue.

BOMA members share your concerns about second-hand smoke in federal buildings. Most Americans spend the majority of their day indoors, and building owners and managers have a responsibility to their tenants to provide and maintain a healthy indoor environment. If the Federal government were to mandate a smoking ban in either public or private workplaces, BOMA would support this mandate as part of an overall effort to improve the quality of indoor air.

A healthy indoor environment is a marketplace concern, important in attracting and retaining tenants. Increased media and legislative attention, however, has caused this to become a higher profile issue in recent years. Reliable information is needed to address this situation effectively and responsibly. BOMA members have long pushed for research on the sources of, and contributors to, indoor air quality problems -- and we are adamant about the need for good guidance.

BOMA International has worked with industry groups and government agencies in developing our efforts on indoor air quality. We have also implemented a strong campaign to distribute sound guidance to improve the indoor air quality management programs in the commercial property community. In cooperation with the EPA and our local associations around the country, we have held a nationwide series of over 50 seminars based on the EPA manual Building Air Quality.

I am pleased and proud to say that, in January, BOMA International's Board of Governors unanimously passed a resolution to support a federal ban on smoking in the workplace -- which I request be included in the record. We see this resolution as a responsible and pragmatic step forward.

Previous legislation has proposed to address indoor air quality problems, including second-hand smoke, by mandating ventilation standards to introduce more outside air into a building. While this may dilute or redistribute polluted air, this does not solve the problem by controlling its source. Since most buildings do not have separate ventilation systems for designated smoking areas, second-hand smoke is distributed into non-smoking areas of the building, endangering the well-being of non-smokers.

Separately ventilated areas for smokers are the only way to ensure that second-hand smoke is not redistributed into other areas of the building. However, most existing buildings are not separately ventilated, and as the General Services Administration testified last month, the

retrofitting of ventilation systems is very costly, if not impossible in some cases. In new buildings alone, the design and installation of separate ventilation systems would cost $30-50 per square foot.

Furthermore, the Environmental Protection Agency has recently classified second-hand smoke as a "Class A" carcinogen. This finding further emphasizes the responsibility of building management to protect the health of office building tenants, their employees, and their guests and clients who may be exposed to this known carcinogen.

BOMA firmly believes that the most effective course of action is to prevent contaminants from being introduced into the workplace in the first place. Second-hand smoke is one of the leading contributors to indoor air pollution, and banning smoking in the workplace would significantly improve the quality of the air we breathe.

We do, however, have several concerns with a federal ban -- first and foremost that its enforcement should not be the responsibility of the nation's building owners and managers. GSA's present policy is that the head of each agency is responsible for implementing smoking policies. In cases where a federal agency leases space in a multi-tenant office building with a building-wide smoking ban, the building manager should not be responsible for "policing" the employees of that agency or the other tenants. Instead, each tenant representative should be responsible for ensuring that the occupants within its space conform to the smoking ban.

Our second concern is that any liability incurred must not apply to the owners and managers of commercial real estate leased to a federal agency when smoking does occur after a ban is imposed. Building management will take the necessary steps to implement a smoking ban and educate tenants on the health risks associated with smoking and second-hand smoke, but cannot take responsibility for building occupants who refuse to comply with the ban.

In conclusion, a uniform federal smoking ban would enable building managers to more effectively control the quality of the indoor environment, through eliminating one of the most harmful sources of indoor air pollution. If a federal ban is imposed, BOMA members will support it, and BOMA International will do everything we can to educate building owners, managers, and tenants to effectively ban smoking in the workplace.

Thank you. I will be pleased to answer any questions.

BUILDING OWNERS AND MANAGERS ASSOCIATION INTERNATIONAL

OFFICE BUILDING OWNERS AND MANAGERS SUPPORT SMOKING BAN

For AMs of
January 22, 1993

Contacts: Henry Chamberlain
Jeanie Markel
(202) 408-2686

WASHINGTON, D.C. --- The Building Owners and Managers Association (BOMA) International voted unanimously at its recently concluded Winter Business Meeting to support a federal ban on smoking in the workplace.

"The Environmental Protection Agency's (EPA) classification of second-hand tobacco smoke as a "Class A" carcinogen leaves no doubt that cigarette smoke must be eliminated from the workplace to ensure public health," said Stephen P. Hokanson, RPA, CPM, President of BOMA International. "A national ban on smoking will greatly improve the indoor air quality of the nation's office buildings."

The resolution stated that BOMA would support a federal ban on smoking in the workplace, as part of an overall effort to improve the indoor environment. In order to be effective, the ban must be a federally-mandated law, making enforcement everyone's responsibility. Once in place, the law will attract widespread support and cooperation for removing smoking from the workplace.

BOMA firmly believes that the most effective way to ensure indoor air quality is to prevent contaminants from being introduced into the workplace in the first place.

- Over -

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BOMA

Smoking Ban - 2

According to an EPA report on the risk of passive cigarette smoke, over 3,000 healthy non-smokers die annually of lung cancer from passive smoke. The Occupational Safety and Health Administration (OSHA) has estimated that up to 77% of the non-smoking workforce, approximately 75 million people, is exposed to passive tobacco smoke while at work.

BOMA International has spent the last year presenting a seminar series, "Improving the Indoor Air Condition," in cooperation with the EPA. BOMA members have long pushed for further research on the sources and causes of indoor air quality problems, testifying on Capitol Hill a number of times to push for more information on identifying sources of IAQ problems.

Founded in 1907, BOMA International is the oldest and largest trade association exclusively representing the office building industry. Its members own or manage over 5 billion square feet of North American office space.

# # #

(A copy of the full resolution is attached.)

RESOLUTION
BEFORE THE
BOMA BOARD OF GOVERNORS
JANUARY 17, 1993
TUCSON, ARIZONA

Whereas, indoor air quality has been the focus of increased media, legislative and regulatory attention for the past several years, with expectations that the intensity of that attention will significantly increase in the months and years ahead.

Whereas, indoor air quality, as a market place demand within the commercial real estate industry, requires no regulation, but requires much information to address this situation effectively and efficiently.

Whereas, BOMA members have long pushed for further research on the sources and causes of indoor air quality problems, and we are adamant about the need for good guidance. We have worked with industry groups, environmental organizations and government officials in coordinating our efforts. We have also demonstrated a strong campaign to develop and distribute sound guidance information to prepare and improve the indoor air quality management programs in the commercial property arena.

Whereas, many legislative and regulatory efforts are underway to dilute or redistribute the sources of indoor air contaminants.

Whereas, BOMA firmly believes that the most effective course of action is to prevent contaminants from being introduced to the workplace in the first place.

Whereas, tobacco smoke has been identified as a leading cause of indoor air contamination, and has generated a great deal of concern within the medical/health community.

Whereas, in light of the EPA's recent classification of second-hand tobacco smoke as a "Class A" carcinogen, BOMA is even more concerned with the consequences to the health and well-being of office tenants, their employees and guests who are exposed to second-hand tobacco smoke in the workplace.

Be It Therefore Resolved, that BOMA would support a federal ban on smoking in the workplace, as part of an overall effort to improve the indoor environment. We do, however, have several concerns with a federal ban - first and foremost among them is that the enforcement of any ban should not be the responsibility of the nation's building owners and managers. The second concern is that any liability incurred must not apply to the owners and managers of commercial, rentable real estate when smoking does occur after a ban is imposed.

BOMA
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STATEMENT OF
PATRICIA W. LATTIMORE
ACTING DIRECTOR, U.S. OFFICE OF PERSONNEL MANAGEMENT

before the

SUBCOMMITTEE ON PUBLIC BUILDINGS AND GROUNDS
COMMITTEE ON PUBLIC WORKS AND TRANSPORTATION
U.S. HOUSE OF REPRESENTATIVES

on

H.R. 881
THE "BAN ON SMOKING IN PUBLIC BUILDINGS ACT"

MARCH 11, 1993

I AM PLEASED TO BE HERE TODAY TO DISCUSS H.R. 881, THE "BAN ON SMOKING IN FEDERAL BUILDINGS ACT." H.R. 881 WOULD BAN SMOKING INSIDE FEDERAL BUILDINGS, BEGINNING 6 MONTHS AFTER THE BILL IS ENACTED. THE OFFICE OF PERSONNEL MANAGEMENT WELCOMES THE SUBCOMMITTEE'S INTEREST IN PROTECTING THE HEALTH OF FEDERAL EMPLOYEES, AS DEMONSTRATED BY THIS HEARING.

LET ME BEGIN BY CLARIFYING OUR ROLE, AND THE ROLE OF FEDERAL AGENCIES, IN HEALTH PROMOTION. FEDERAL AGENCIES ARE GIVEN STATUTORY AUTHORITY TO ESTABLISH HEALTH SERVICE PROGRAMS TO PROMOTE AND MAINTAIN THE PHYSICAL AND MENTAL FITNESS OF EMPLOYEES. THE OFFICE OF PERSONNEL MANAGEMENT PROVIDES POLICY GUIDANCE AND TECHNICAL ASSISTANCE TO AGENCIES INTERESTED IN ESTABLISHING PREVENTIVE HEALTH SERVICE PROGRAMS. AS PART OF THIS EFFORT, OPM HAS LONG BEEN CONCERNED ABOUT THE ADVERSE EFFECTS OF SMOKING AND OF SECONDHAND AND SIDESTREAM SMOKE. OVER THE PAST SEVERAL YEARS, OPM HAS ACTIVELY PROMOTED EFFORTS BY FEDERAL AGENCIES TO ENCOURAGE

THEIR EMPLOYEES TO STOP SMOKING.

AS LONG AGO AS 1986, WE BEGAN OUR EFFORTS WITH A CONFERENCE ON SMOKING CESSATION HERE IN WASHINGTON. THE CONFERENCE WAS DESIGNED TO SHOW AGENCIES HOW TO ESTABLISH EDUCATIONAL PROGRAMS ABOUT THE EFFECTS OF SMOKING AND HOW TO ENCOURAGE THEIR EMPLOYEES TO STOP SMOKING. WE HAVE MADE AVAILABLE TO AGENCIES A VARIETY OF EDUCATIONAL MATERIALS ABOUT SMOKING CESSATION AND THE EFFECTS OF SMOKING, AND WE WILL CONTINUE TO DO SO. SINCE FEBRUARY OF 1989, FEDERAL AGENCIES HAVE BEEN AUTHORIZED TO USE AVAILABLE APPROPRIATED FUNDS TO PAY FOR SMOKING CESSATION PROGRAMS FOR EMPLOYEES.

TWO YEARS AGO, WE REQUIRED FEE-FOR-SERVICE PLANS PARTICIPATING IN THE FEHBP -- THE FEDERAL EMPLOYEES' HEALTH BENEFITS PROGRAM -- TO PROVIDE SMOKING CESSATION ASSISTANCE. THIS REQUIREMENT REACHES THE BULK OF OUR WORK FORCE, SINCE 72 PERCENT OF FEDERAL EMPLOYEES ARE COVERED BY FEE-FOR-SERVICE PLANS IN FEHB. HEALTH MAINTENANCE ORGANIZATIONS, THOUGH NOT REQUIRED TO DO SO, USUALLY PROVIDE THEIR OWN SMOKING CESSATION ASSISTANCE SERVICES.

THE RECENT REPORT OF THE EPA, REGARDING THE DANGERS OF ENVIRONMENTAL TOBACCO SMOKE, PROVIDES NEW GROUNDS FOR CONCERN. EVERY EMPLOYER MUST TAKE THE EPA FINDINGS SERIOUSLY. THE REPORT CONCLUDES THAT ENVIRONMENTAL TOBACCO SMOKE IS RESPONSIBLE FOR APPROXIMATELY 3,000 LUNG CANCER DEATHS EACH YEAR IN NONSMOKING ADULTS IN THE UNITED STATES. IT ALSO HAS OTHER SIGNIFICANT EFFECTS

ON RESPIRATORY HEALTH.

THE FEDERAL GOVERNMENT, AS AN EMPLOYER, HAS A CLEAR RESPONSIBILITY TO PROTECT NON-SMOKERS FROM THIS UNDENIABLY SERIOUS HEALTH HAZARD IN THE WORKPLACE. THE OFFICE OF PERSONNEL MANAGEMENT IS COMMITTED TO MEETING THIS RESPONSIBILITY. THEREFORE, WE STRONGLY SUPPORT THE OBJECTIVES OF LEGISLATION SUCH AS H.R. 881 TO PROTECT NON-SMOKERS IN FEDERAL BUILDINGS. WE DEFER TO GSA AND OTHER INTERESTED AGENCIES REGARDING THE DETAILS OF H.R. 881.

AGAIN, I THANK THE SUBCOMMITTEE FOR ADDRESSING THIS ISSUE, AND I WOULD BE HAPPY TO ANSWER ANY QUESTIONS YOU MAY HAVE.

HOUSE PUBLIC WORKS AND TRANSPORTATION COMMITTEE

SUBCOMMITTEE ON PUBLIC BUILDINGS AND GROUNDS

STATEMENT OF MAURICE E. LEVOIS, PH.D.

ON H.R. 881

My name is Dr. Maurice LeVois. I have been asked by the Tobacco Institute to prepare comments on health issues related to exposure to environmental tobacco smoke (ETS) and its possible relationship to lung cancer.

Background and qualifications of the author:

I am the principal scientist in Environmental Health Resources, an association of consulting epidemiologists and statisticians with offices in Mill Valley, California. I received my Ph.D. from the University of California, San Francisco, in 1984. I was formerly director of the Veterans Administration's Office of Agent Orange Research and Education, and a scientist in the Agent Orange Study Unit, Centers for Disease Control. My professional activities have involved the design and conduct of large cohort studies and occupational mortality studies, survey research on problems of artifact in epidemiologic methods, research on cancer incidence and reproductive health effects in populations exposed to agricultural chemicals, and epidemiologic modeling and failure analysis of toxic waste management facilities.

I. ETS in the workplace

It is clear that the proposal to ban smoking in federal buildings is a response to the Environmental Protection Agency's risk assessment on environmental tobacco smoke. I have commented extensively on the EPA report during its review process, and I believe that the conclusions of the report are not scientifically defensible. My discussion of the weaknesses of

the scientific data that were relied upon by the EPA is contained in my written statement as well as in my testimony today. I would like to emphasize the weakness of relying on the EPA report to address whether smoking should be banned in the workplace.

o First of all, the reported association between spousal smoking and lung cancer could easily be explained by factors other than ETS that are known to be related to spousal smoking. The EPA incorrectly attributes the entire spousal smoking / lung cancer association solely to ETS instead of the whole complex of dietary, health history, environmental, occupational, and lifestyle factors that constitute the spousal smoking variable–including the known positive bias due to misclassification of the smoking status of study subjects.

o In any event, there are enough data, from 13 epidemiologic studies of ETS exposure in the workplace, to do the same sort of "meta-analysis", or pooling of results, that EPA used in their risk analysis of the ETS studies relating to women in the home. The summary relative risk for the workplace ETS studies is 1.01 and is statistically not significant – i.e. no association between ETS exposure and lung cancer in the pooled workplace data.

This may be because the workplace data are not subject to the many biases and confounders that are an integral part of the spousal smoking definition.

This inconsistency undermines both EPA's interpretation of the spousal smoking studies and the claim that workers who are exposed to ETS are at an increased risk of lung cancer.

o It is important to keep in mind that when we talk about exposure to ETS constituents in real world situations we are talking about extremely low levels of exposure, levels well below the permissible exposure limits set by OSHA. For example, it would be necessary to burn 135,000 cigarettes in a 3'X3'X3' box in order to reach the benzo[a]pyrene levels used to dose hamsters in laboratory experiments (9500 ug/ m^3). Benzo[a]pyrene, benzene, nickel, nitrosamines, and other constituents reported in sidestream smoke are present at extremely low levels in nearly all indoor and outdoor environments. There is no consensus that such low exposures are harmful.

o The following is a list of the approximate number of cigarettes that would have to be simultaneously burned in a small sealed non-ventilated office (10' X 10' X 10') before reaching the Threshold Limit Value (TLV) for occupational settings for several SS constituents:

Constituent	Cigarettes	Constituent	Cigarettes
Cadmium	1400	benzo[a]pyrene	285,000
Acetaldehyde	1400	2-Toluidine	300,000
Benzene	6250	Polonium-210	2,500,000
Nickel	40,000		

o There are trace levels of carcinogens generally present in the environment, both indoors and outdoors. Many of them are present at far higher levels than found in ETS and yet they are not banned by regulatory agencies. While some would interpret theoretical

models of carcinogenesis in such a way that even a single molecule of a carcinogen is viewed as a threat, this type of fear is unrealistic and counter productive. It is all the more so when the fear is narrowly focused on one constituent among hundreds.

o Eliminating ETS from indoor environments would not address the many other unwanted constituents in indoor air that sometimes reach relatively high levels of concentration, such as products that result from cooking, heating, office machines, building materials, microbes, etc. On the other hand, improved ventilation would lower the levels of all unwanted indoor air constituents. Whether the priorities are based upon health or comfort considerations, banning smoking indoors is not the appropriate solution for improving indoor air quality.

II. Weaknesses in the ETS epidemiology

The presence of very low concentrations of environmental tobacco smoke (ETS), volatile organic compounds, asbestos, and traces of many other indoor air constituents, often found even in properly ventilated buildings, has not been scientifically established to pose a risk of disease. However, the fear of toxic chemicals is very high among the general public, and exaggerating the risk of health effects due to very low level chemical exposures provides fuel for that fear. Indoor air quality standards should take dose into account and reflect the best scientific understanding of whether very low level exposures pose real health risks, otherwise even greater public fear and confusion are likely to result.

Environmental tobacco smoke is a case in point. Some of the epidemiologic studies that have used spousal smoking as a proxy for ETS exposure have reported weak associations between spousal smoking and various disease endpoints. However, such studies are highly vulnerable to bias and confounding due to the concordance of spouses with respect to numerous health risk factors that are correlated with living with a smoker. Such spousal smoking studies are not directly relevant to the occupational setting. However, the relationship between lung cancer mortality and exposure to ETS in the workplace has now been investigated in a fifteen epidemiologic studies, and the summary relative risk for those studies is 1.01 (i.e. ETS has not been found to be related to the risk of lung cancer in the workplace). It is noteworthy that epidemiologic studies of ETS exposure in the workplace are unlikely to be influenced by artifact that operates through spousal concordance.

The level of ETS found in the homes of smokers, about 20-50 micrograms/ m^3 RSP (respirable suspended particulates), is similar to or higher than that found in smoking areas of most properly ventilated buildings, and is orders of magnitude less than permissible exposure levels set by the Occupational Safety and Health Administration (OSHA) for many chemicals designated to be carcinogenic. It would make no sense to regulate ETS more strictly than any other substance on the basis of unsubstantiated health effects claims. To do so would send the wrong message to the public about what constitutes a demonstrated environmental health risk.

There are important weaknesses and inconsistencies in the epidemiologic data which undermine the inference that ETS causes lung cancer. The main points of criticism are as follows:

1. The ETS epidemiologic data are very weak and inconsistent. The magnitude of the reported association between ETS and lung cancer is in the range of relative risk 1.0-1.3. Not only is this association less than most epidemiologists feel comfortable interpreting even under the best of circumstances (relative risks of about 2.0-3.0 are frequently mentioned as the minimum epidemiologic relative risk that can be interpreted with a reasonable degree of confidence), the studies themselves are very weak in both their design and conduct. For example, questionnaire data on spousal smoking is the main, indirect, exposure measure in all of the epidemiologic studies, introducing clear potential for both bias and confounding in all of the studies. Most of the studies employ questionnaire data obtained from surrogate respondents despite evidence presented in the large U.S. study conducted by Garfinkel et al.(1985) that this is a source of positive bias. Few of the studies employ data on specific lung cancer cell types, and the results of those that do are remarkably inconsistent and even conflicting.

2. Meta-analysis does not support a causal inference. Meta-analysis, that is, pooling data from all of the ETS epidemiologic studies, has been relied upon extensively by policy makers who have reviewed the ETS data, but this approach provides questionable support for a causal inference. Inconsistencies among the studies, heterogeneity in study results, design flaws that make all of the ETS studies individually susceptible to bias and

confounding, and the recognized problem of publication and file-drawer bias, all weaken the applicability of meta-analysis. The introduction of global adjustments to the data, based upon speculation and circular arguments, that are included in the major ETS policy reviews (e.g. "background exposure" adjustments) is simply unscientific.

3. Inconsistent dose-response results. The ETS epidemiologic studies provide weak and inconsistent evidence of a lung cancer dose-response relationship at best, with the largest and best executed U.S. studies reporting little sign of a possible dose-response effect.

It should be noted that weak spurious trends in association, such as those claimed in some ETS epidemiologic studies, may result if an uncontrolled bias or confounder is correlated with ETS exposure. For example, dietary deficiencies related to lung cancer risk are known to be similar for both smokers and their nonsmoking spouses (Friedman et al., 1983; Koo et al., 1988; Koo, 1989). Such deficiencies are correlated with both lung cancer risk and spousal smoking behavior–they tend to get worse as the level of active smoking increases.

The trend analysis methods used in all of the ETS epidemiologic studies (improperly called "dose-response" analyses in some of the papers) ignores such known correlates of both ETS exposure and lung cancer risk as active smoker misclassification and confounders such as diet, socioeconomic status, and high risk occupational exposure. Because there is often a large initial difference in exposure to confounders separating subjects who are "exposed" and "not exposed" to some other study factor, such as ETS, Breslow and Day (1987) recommend conducting dose-response analysis only within the "exposed" group. This approach can not completely eliminate the risk of misinterpreting the effects of correlated biases or

confounders, but it at least protects against obvious artifactual differences between "exposed" and "not exposed" groups. Not one of the ETS epidemiologic studies has used the recommended method for conducting a proper dose-response analysis.

4. **Inconsistent dosimetric prediction.** At the present time both the ETS epidemiologic and dosimetric measures are too unreliable to predict risk with an acceptable degree of certainty. However, in light of the fundamental flaws in the ETS epidemiologic studies, the dosimetric approach to estimating risk is certainly worth considering. It has the advantage of more clearly specifying the assumptions employed and the degree of uncertainty involved. As seen in Table 1, the level of lung cancer risk predicted from a combination of active smoking epidemiologic and ETS dosimetric data is two to three orders of magnitude lower than the ETS epidemiologic estimate (depending on the choice of assumptions and constituents). This discrepancy challenges the biological plausibility and the validity of the associations reported in the ETS epidemiologic studies.

Table 1

Extrapolated Relative Risk Based on Cigarette Equivalents

Author	Cigarette equivalent	Lung Cancer Relative Risk: Exposed ages 20-65 years	Lung Cancer Relative Risk: Exposed ages 0-65 years
Adlkofer et al. '89	0.005-0.012	1.002-1.006	1.004-1.009
Arundel et al. '87	0.0035*	1.002	1.003
McAughey et al. '89	0.005	1.002	1.004
Robins et al. '89	0.0003-0.015	1.0001-1.007	1.0001-1.012

* Estimate for females

5. Inappropriate assumptions about the claimed carcinogenicity of ETS. Dosimetric estimates of ETS lung cancer risk are rejected by some in part because of speculation that ETS may be more toxic than mainstream smoke (MS). There are no convincing data to support such speculation. The relative concentrations of sidestream smoke (SS) and MS constituents are irrelevant to this issue. ETS is so highly diluted that the primary respiratory defense mechanisms of ETS-exposed individuals can be expected to reduce appreciably any respirable suspended particulate (RSP) dose delivered to the lungs.

6. Cancer trends do not reflect any ETS effect. If, as some have claimed, ETS exposure causes more than a fourth of all female nonsmoker lung cancer deaths in the U.S., then annual female nonsmoker lung cancer incidence rates over the past four decades should have followed the rise and fall of prevalence rates for male smoking. Actual female nonsmoker lung cancer rates, however, do not reflect such a linkage (Wynder and Kabat, 1990; Garfinkel and Silverberg 1990). Such basic and discrepant epidemiologic data seriously undermine the biological plausibility of an ETS-related lung cancer risk.

7. Inconsistent cell types. The association of active smoking with adenocarcinoma that has been reported in the literature on females is less than half that reported for squamous cell carcinoma (Shimizu et al., 1982). However, the opposite relationship is reported in most of the ETS epidemiologic studies that have looked at risk by cell type, with adenocarcinoma being more highly associated with spousal smoking than are other cell types. Current clinical practice for diagnosing lung cancer does not provide for a clear distinction between primary and metastatic lung cancer, which according to Faccini (1989) raises the possibility that many of the adenocarcinomas of the lung that comprise a majority of cases in the ETS epidemiologic studies are secondary to cancer of the breast, bowel, ovary and uterus, all common cancer sites where adenocarcinoma predominates. ETS epidemiologic studies of lung cancer in females may, therefore, reflect the effects of ETS-related lifestyle confounders that are important risk factors for these other common cancers in females.

A further discrepancy exists between the cellular characteristics reportedly associated with ETS exposure and what is expected based upon current understanding of lung cancer etiology.

Damage to the bronchial epithelium is believed to be necessary before deposition of carcinogenic particles can have an effect (Wynder and Kabat, 1990). Although it has been claimed that ETS exposure is associated with epithelial damage (Trichopoulos et al., 1992), the data presented in that paper do not even indicate a difference between heavy active smokers and nonsmokers, let alone an epithelial effect of ETS exposure. In fact, it has been observed by others that damage to bronchial epithelium is extremely rare in lifetime nonsmokers (Wynder and Kabat, 1990; Auerbach et al. 1961).

In 1987, after reviewing the 12 epidemiologic studies then available, the International Agency for Research on Cancer (IARC) concluded that spousal smoking epidemiologic studies were too flawed to interpret, noting that no spousal smoking study adequately accounted for potential sources of bias and confounding. The ETS epidemiologic studies that have appeared since 1987 employ exactly the same flawed spousal smoking study design, and continue to either disregard potential sources of bias and confounding altogether, or fail to address the problems adequately. The epidemiologic data do not convincingly support the inference that ETS causes lung cancer.

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Third District, Kentucky

NEWS RELEASE

Congressman Ron Mazzoli

Testimony of the Honorable Romano L. Mazzoli
House Subcommittee on Public Buildings and Grounds
Smoking in Federal Buildings
March 11, 1993

Mr. Chairman:

Thank you very much for allowing me to appear before your subcommittee today with my distinguished colleague from Illinois, Mr. Durbin. It is also an honor to testify after the Surgeon General of the United States, Dr. Antonia Novello who has been a true leader on smoking and health issues. In particular, I recall her efforts last year to ask the Tobacco industry to voluntarily halt the Joe Camel advertising campaign, an effort which I also took up on the Floor of the House.

The bill before the subcommittee today is H.R. 881, legislation which would prohibit smoking in federal buildings, including the United States Capitol. As you are probably aware, I, along with my colleagues, Mr. Durbin and Mr. Hansen of Utah, am a sponsor of H.R 710 introduced in early February. Our bill, the PRO-KIDS Act of 1993, would also establish a no-smoking policy in the federal workplace and in any federally funded facilities housing children.

Mr. Chairman, as I said on the Floor a few weeks ago, the trend today is toward smoke-free facilities. The White House has gone smoke-free. Governor Wilson in California has signed an executive order prohibiting smoking in state buildings, prisons, and hospitals. Governor Brereton Jones in my own state of Kentucky is implementing a smoking policy that would limit smoking to designated areas in state buildings. And, Louisville city government and Jefferson County government agencies are also going this route.

This comes about in large part because of the mounting evidence that tobacco smoke contains ingredients harmful to both the user and to those who breathe smoke in secondhand.

The Environmental Protection Agency report of January 7, 1993 is the latest and among the more persuasive such statements. The report classified secondhand smoke as a Class A carcinogen and identified it as responsible for approximately 3000 lung cancer deaths annually in nonsmokers. The report also concluded that exposure to secondhand smoke causes hundreds of thousands respiratory illnesses among children and increases the severity of asthma among children who have that affliction.

Some groups are challenging the science behind the EPA's report, but the plain fact is this: exposure to tobacco smoke -- either directly or passively inhaled -- cannot be good for anyone's health, and may in fact be quite harmful to one's health. For this reason, and for the legal liability issues raised by the EPA report, governments and private firms throughout the country are moving to restrict and even prohibit smoking in their buildings. And, I do not doubt that the Occupational Safety and Health Administration (OSHA) is examining workplace rules changes relating to smoking at this very moment.

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The time has come for the Federal government to adopt a non-smoking policy to promote the health of both smokers and non-smokers. And, this policy should cover the Capitol complex. I would note that a recent survey conducted by Mr. Durbin concluded that many Members of the House support making the House smoke-free.

Finally, although I come from a tobacco state, I am taking this position because of my concern for the health of all people. And, in the coming debate over health care reform, tobacco use, and tobacco taxes, Congress has an obligation to establish a conversion program for tobacco farmers and workers in tobacco dependent states like Kentucky.

This means creating transition and conversion programs to assist tobacco growers in moving into alternative crops, helping tobacco states attract food processing facilities to give growers markets for produce other than tobacco, and providing retraining assistance for tobacco workers who might be displaced due to decreasing tobacco production.

Those good people in my state who -- for reasons of tradition and history -- have made their living from tobacco will be affected by these national trends and the actions of government and, as in the case of defense worker-conversion programs, the government has a responsibility to help these people.

So in conclusion, I support your efforts and I thank you again for allowing me to testify.

I welcome your questions at this time.

DEPARTMENT OF HEALTH & HUMAN SERVICES

The Surgeon General of the
Public Health Service
Washington DC 20201

STATEMENT OF

ANTONIA C. NOVELLO

SURGEON GENERAL

PUBLIC HEALTH SERVICE

DEPARTMENT OF HEALTH AND HUMAN SERVICES

BEFORE THE

SUBCOMMITTEE ON PUBLIC BUILDINGS AND GROUNDS

COMMITTEE ON PUBLIC WORKS AND TRANSPORTATION

U.S. HOUSE OF REPRESENTATIVES

MARCH 11, 1993

Mr. Chairman and Members of the Subcommittee:

I am Dr. Antonia C. Novello, Surgeon General of the United States Public Health Service, Department of Health and Human Services. As the Subcommittee requested, I will present testimony on the effects of smoking and, in particular, the effects of secondary smoke on nonsmokers. As a public health official, physician, and Federal employee, I commend the Subcommittee for addressing exposure to environmental tobacco smoke as an important Federal workplace health and safety concern. I appreciate the opportunity to summarize the deadly health consequences of smoking.

HEALTH EFFECTS OF SMOKING

Cigarette smoking is the single most preventable cause of disease and premature death in the Nation. The magnitude of the adverse health and economic consequences of smoking are enormous. Smoking affects every one of us: We know someone who smokes, and who will become ill or <u>die</u> as a result of smoking. We also are affected by breathing environmental tobacco smoke -- known to cause lung cancer and other disease (to say nothing of discomfort).

Most significantly affected, however, are the 45.8 million adult smokers in the United States -- many of whom will suffer a smoking-related illness, disease or death. Smoking causes or contributes to numerous diseases. It is a major risk factor for cancers of the lung, larynx, pharynx, oral cavity, esophagus; for

diseases of the heart and blood vessels; chronic obstructive pulmonary disease, chronic bronchitis and emphysema; and peptic ulcer disease. Smoking is considered to be a contributing factor for cancers of the kidney and bladder.

Ultimately, 434,000 Americans die each year because of smoking. This represents 1 out of every 5 deaths, and includes 21 percent of all coronary heart disease deaths, 87 percent of lung cancer deaths, and 30 percent of all cancer deaths. Many smoking-related deaths occur before age 65, striking people in the prime of their life.

Death rates alone understate the public health impact of tobacco use. Smoking contributes substantially to chronic illness and disability as well. The estimated cost of smoking-attributable health care and related services is more than $65 billion dollars a year. On average, smokers miss eight more days of work a year than do nonsmokers. Absentee rates average 30 to 50 percent higher among smokers than nonsmokers. Every employee who smokes costs his or her company, on average, at least $1,000 a year because of decreased productivity and increased health care costs. The intangible costs of smoking - in terms of human suffering - are immeasurable.

In addition to being a causal or contributing factor in disease and premature death, smoking itself now is recognized as a

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disease, known as nicotine addiction. The World Health Organization and other medical and health entities officially have classified nicotine addiction as a treatable disease.

Smoking also has effects that go beyond the individual smoker. One example is maternal smoking, especially during pregnancy. Maternal smoking during pregnancy retards fetal growth and is associated with an increased incidence of low birth weight, premature birth, miscarriage, stillbirth, sudden infant death syndrome, and infant mortality. In the United States, 20 to 30 percent of the incidence of low birth weight and up to 14 percent of pre-term deliveries are attributable to maternal smoking during pregnancy.

HEALTH EFFECTS OF EXPOSURE TO ENVIRONMENTAL TOBACCO SMOKE

Although tobacco smoking long has been recognized as a major cause of disease and premature death for smokers, a growing body of scientific evidence in recent years has documented that non-smokers also are at risk for adverse health effects as a result of their exposure to environmental tobacco smoke, or ETS. ETS is the combination of the smoke exhaled by smokers and the smoke given off by the burning end of cigarettes. Exposure to environmental tobacco smoke often is referred to as "passive smoking" or "secondhand smoke."

Passive smoking is well-known and widely recognized as a cause of

headache, nausea, watery eyes, runny noses, and other short-term effects. In addition, we have recognized passive smoking as a cause of acute illness in people with special sensitivity to tobacco smoke, such as those with allergies, or asthma, or emphysema.

In 1986, the U.S. Surgeon General and the National Research Council independently assessed the health effects of exposure to ETS. Both concluded that ETS can cause disease, including lung cancer, in non-smokers. These reports also substantiated that children of parents who smoke have an increased frequency of respiratory symptoms and lower respiratory tract infections. Furthermore, the Surgeon General concluded that the simple separation of smokers and nonsmokers within the same air space may reduce, but does not eliminate, nonsmokers' exposure to tobacco smoke.

Since 1986, the number of studies which examine these issues in human populations has more than doubled, resulting in a large body of knowledge with which to conduct comprehensive assessments of the potential effects of passive smoking on the respiratory health of adults and children.

For example, the National Institute for Occupational Safety and Health (NIOSH), within the HHS Centers for Disease Control and Prevention, has assessed environmental tobacco smoke in the

workplace. In 1991, in its official analysis, "Environmental Tobacco Smoke in the Workplace: Lung Cancer and Other Health Effects," NIOSH determined that ETS meets the criteria for classification as a potential occupational carcinogen. In order to protect workers, NIOSH recommended that ETS exposure be reduced to the lowest feasible concentration, and that all preventive measures be used to minimize occupational exposure. NIOSH concluded that worker exposure to ETS is most efficiently and completely controlled by simply eliminating smoking from the workplace.

More recently, the Environmental Protection Agency (EPA) undertook a scientific risk assessment of respiratory effects of exposure to environmental tobacco smoke. Released in January 1993, this assessment builds on earlier reports and was based on a thorough review of all of the studies in the available literature. Consistent with earlier reports, EPA documented that environmental tobacco smoke exposure is both a cause of lung cancer in adults and an especially significant health risk for children.

Based on the weight of available scientific evidence, EPA officially has classified environmental tobacco smoke as a Group A (known human) carcinogen under EPA's carcinogen assessment guidelines. EPA reserves this classification exclusively for those compounds or mixtures which have been shown to cause cancer

in humans, based on studies in human populations.

The widespread exposure to environmental tobacco smoke in the U.S. presents a serious and substantial public health risk. Exposure to environmental tobacco smoke is responsible for approximately 3,000 lung cancer deaths among adult nonsmokers annually in the United States. Adults exposed to environmental tobacco smoke in the work environment are also at greater risk of developing small airways dysfunction than are nonexposed employees. Small airways disease, which is the first pathological change seen in beginning smokers, may increase the risk of developing disabling chronic airways obstruction.

Because of the role of this Subcommittee in the Federal workplace, adult workers are a primary area of interest. Because Federal facilities serve or are open to children, however, I also would like to summarize the effects of secondhand smoke on children.

In children, there is a causal association between exposure to environmental tobacco smoke and respiratory effects including increased risk of lower respiratory tract infections (bronchitis and pneumonia), increased symptoms of irritation (cough and wheeze), increased episodes and increased severity of symptoms in children with asthma, and a small but significant reduction in lung function. Environmental tobacco smoke exposure also

increases the prevalence of fluid in the middle ear in children, a sign of chronic middle ear disease. Secondhand smoke also is a risk factor for new cases of asthma in children who have not previously displayed symptoms.

The Department of Health and Human Services recently joined with hundreds of public health officials and organizations throughout the Nation to develop health objectives for the Year 2000. This blueprint for improving health in our Nation includes numerous objectives regarding tobacco use. Of special relevance for this Subcommittee is that prohibiting or strictly limiting smoking in workplaces and enclosed public places is one of our country's significant and achievable health goals.

CONCLUSION

The health effects of smoking and passive smoking are <u>preventable</u> -- which means that something can be done to eliminate them. The scientific case against environmental tobacco smoke justifies - and should compel - action to protect people, especially children, from the potential health effects of exposure to environmental tobacco smoke. To reduce health risks, restrictions to prevent exposure <u>must</u> be more comprehensive than those currently in place in most facilities.

In closing, let me restate that the general direction of H.R. 881 would provide important public health safeguards for those who work in and visit Federal buildings. The Department of Health and Human Services supports the objectives of H.R. 881.

By dealing with this health issue, this Subcommittee has assumed an important leadership role in public health. I commend you for addressing one of the most important, sensitive, and far-reaching workplace health issues of our time, and for considering measures to protect those who work in or visit Federal facilities throughout the country.

Mr. Chairman and members of the Subcommittee, this concludes my formal statement. I would be happy to answer any questions that you and other members of the Subcommittee might have.

Department of Health and Human Services

OFFICE OF INSPECTOR GENERAL

SPIT TOBACCO AND YOUTH

OEI 06-92-00500

EXECUTIVE SUMMARY

PURPOSE

This study describes the present status of youth use of spit tobacco in six areas: prevalence and patterns of use, health effects, environmental influences on use, product promotion and sales, regulation and enforcement, and educational efforts.

BACKGROUND

This report, requested by the Surgeon General, is the second on spit tobacco (snuff and chewing tobacco) by the Office of Inspector General. The first, in 1986, concluded spit tobacco use by young people was a national problem with serious potential health consequences. The current inspection focused on data developed after 1985 for users under age 19 and found spit tobacco use persists as a national health problem. Information came from four major sources: literature review, national and regional data on prevalence and use patterns, experts in spit tobacco issues, and young users.

FINDINGS

Under-Age Spit Tobacco Use Is High, Wide-Spread And Begun Early.

In 1990 and 1991, nearly one in five high school males used spit tobacco. However, high spit tobacco use is not confined to one region or group; northeasterners, urban dwellers, and Native American children use spit tobacco. Age 11 or 12 is typical for starting use.

Often Addicted To Spit Tobacco, Under-Age Users Are Seriously Endangering Their Health.

Spit tobacco contains cancer-causing and addictive substances. Short term spit tobacco use often causes leukoplakia (white, wrinkled, skin patches inside the mouth) which can, over time, become malignant in a significant percentage of cases. Long term use greatly increases the risk for oral cancer which may become epidemic if young people continue frequent use into their adult years. Use is intense among young people who frequently admit they are addicted to nicotine.

Family, Friends And Other Incentives Strongly Contribute To Under-Age Use.

Perceived social support from fathers, other male relatives and friends is the most influential reason youth first try spit tobacco. The baseball connection, product placement in stores and "look-alike" items further enhance acceptability of use.

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INTRODUCTION

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This study describes the present status of youth use of spit tobacco in six areas: prevalence and patterns of use, health effects, environmental influences on use, product promotion and sales, regulation and enforcement, and educational efforts.

BACKGROUND

Spit tobacco includes two main types: snuff and chewing tobacco. Snuff "dippers" place a small amount of shredded or finely ground tobacco (loose or encased in a paper pouch) between their cheek and gum. Tobacco "chewers" place a wad of loose leaf tobacco or a plug of compressed tobacco in their cheek. Both dippers and chewers suck on the tobacco and spit out the tobacco juices and saliva generated. Previously termed "smokeless tobacco", snuff and chewing tobacco are now sometimes called "spit" or "spitting" tobacco to more accurately describe how they are used and to counteract the misconception that they are safe to use. This study, requested by the Surgeon General, uses the newer nomenclature of spit tobacco.

Youth use of spit tobacco is a serious public health concern. In January 1986 the OIG released a national study entitled, "Youth Use of Smokeless Tobacco: More Than a Pinch of Trouble." This report concluded that increasing use of smokeless (spit) tobacco by young people was a national problem with serious potential health consequences. Young users were already experiencing health effects and were unaware of the risk for others. Subsequently, offices within the Public Health Service (PHS), i.e., the Centers for Disease Control (CDC), National Cancer Institute (NCI), National Institute of Dental Research (NIDR), National Institute on Drug Abuse (NIDA), Office of the Surgeon General, and the Indian Health Service, compiled a growing body of completed and developing scientific research information that shows youth use of spit tobacco persists as a national health problem. The American Dental Association and the American Academy of Otolaryngology, among others, promote practices that actively seek to deter youth use.

In 1986, to deter spit tobacco use, Congress enacted the Comprehensive Smokeless Tobacco Health Education Act (CSTHEA) of 1986 (P.L. 99-252). The Act required the Secretary to inform the public of any health dangers of smokeless (spit) tobacco use. Smokeless (spit) tobacco packaging and advertising had to include warning labels with the exception of outdoor billboard advertising. Advertising on radio or television was banned. The Act also encouraged States to establish 18 as the minimum age for the purchase of smokeless (spit) tobacco, currently the standard in all but three States. In July 1992, the Congress further strengthened national law when it passed the ADAMHA Reorganization Act which required all States to ban the sale and distribution of tobacco products to everyone under age 18 by October 1, 1994. It also requires States to enforce their laws

FINDINGS

Under-Age Spit Tobacco Use Is High, Wide-Spread And Begun Early.

- ***Currently, nearly 1 in 5 high school males uses spit tobacco; some State rates are considerably higher.***

Percent use during preceding 30 days
Grades 9 to 12

	Males		Females	
Year	White	Total	White	Total
1990	23.9%	19.1%	1.5%	1.4%
1991	23.6%	19.2%	1.4%	1.3%

Source: YRBS, 1990 (N = 11,631) & 1991 (N = 12,272)

Table 1

A 1990 and 1991 school-based survey in 22 select States (Youth Risk Behavior Survey - YRBS) shows that nearly 20 percent of males in grades 9 to 12 were current users of spit tobacco (1-3).[a] Among white males, an even higher 1 in 4 currently use spit tobacco (see Table 1). Use among white male 12th graders in 1990 was 26 percent. Rates for Hispanic and Black teens are notably lower, as are female use rates.

In some States, current use by high school males is much higher than the composite YRBS rate. Overall male usage rates in grades 9 to 12 are as high as 34 percent in Tennessee and 33 percent in Montana (3). Other States with high overall prevalence among high school males are Colorado (32%), Alabama (31%) and Wyoming (31%) (2). Further, in States with high use rates, more 9th or 10th grade males than senior males may currently use spit tobacco (see Table 2). For additional States with high spit tobacco use rates, see Appendix B.

Current Use By Grade

Grade	Alabama	Tennessee
9th	36.6%	35.2%
10th	26.7%	37.1%
11th	26.9%	30.7%
12th	31.1%	33.7%

Source: YRBS 1991 (109, 111)

Table 2

- ***Spit tobacco use by young males has increased greatly since 1970 and has stayed high.***

Between 1970 and 1985 spit tobacco use by young males increased dramatically nation wide. The 1970 National Health Interview Survey reported young males age 17 to 19 were the lowest snuff users while men age 50 and over were the highest (4). By 1985, the Current Population Survey showed males age 16 to 19 had a snuff usage rate double that of men age 50 and over.[b,c]

Other studies showed high spit tobacco use among young people. In 1989 the NCAA replicated its 1985 national study of social drug use by college athletes (5). It found use of spit tobacco by college athletes had risen from 20 percent in 1985 to 28 percent in 1989, a 40 percent increase. More importantly, the NCAA found the large majority of college users began dipping or chewing before they went to college, i.e., either in junior high (21%) or senior high (54%) school. Furthermore, spit tobacco was the only social

old or younger. Twenty-eight percent (15 of 54) of them were five years old or younger when they first tried spit tobacco.

In some regions, regular use also occurs at early ages. For example, 17 percent of West Virginia's 5th and 6th grade boys are regular spit tobacco users (19b). Other States are experiencing a smaller, but significant, proportion of their young children using spit tobacco regularly: 3.7 percent of Illinois's 5th graders (11), 3 percent of Oklahoma's (20) and Missouri's 6th graders (12), and one percent of Maine's (8) and Connecticut's 5th graders (7). In 1986 we reported the average age for regular or daily use was 12.

Often Addicted To Spit Tobacco, Under-Age Users Seriously Endanger Their Health.

- ***Spit tobacco contains cancer-causing and addictive substances.***

Spit tobacco contains tobacco nitrosamines and other proven carcinogens, i.e. cancer-causing substances (21). Spit tobacco nitrosamine levels are hundreds, or in some cases thousands, of times greater than what foods and beverages may legally contain (4,22). For example, spit tobacco has 100 times more nitrosamines than bacon (23). Moist snuff has a much higher nitrosamine level than looseleaf chewing tobacco. While some researchers have suggested setting maximum permissible levels of nitrosamines (21), NCI has said nitrosamines are not safe at any level in tobacco products (24). Pinkerton's Red Man moist snuff has the highest nitrosamine level of any snuff sold in the United States, followed by U.S. Tobacco Company's (UST) Copenhagen (21,25,26). Moist snuff also contains polonium-210, uranium-235 and -238, nickel, and formaldehyde, also considered carcinogens.

Another harmful ingredient of spit tobacco is nicotine, an addictive drug (27). Nicotine enters the user's bloodstream by being absorbed through the lining of the mouth. In normal use, a snuff dipper places a pinch in his mouth and holds it there for 20 to 30 minutes. The amount of nicotine absorbed is 2 to 3 times the amount delivered by a regular-size cigarette (25). Chewing tobacco has less available nicotine (per gram of tobacco) compared to moist snuff, but chewers use more tobacco per dose. Thus, users who consume 8 to 10 dips or chews a day receive a nicotine dose equal to that taken by a heavy smoker who consumes 30 to 40 cigarettes daily (25). Nicotine is absorbed more slowly from spit tobacco than from cigarette smoke. However, more nicotine per dose is absorbed from spit tobacco and it stays in the bloodstream longer (28). UST's Copenhagen and Pinkerton's Red Man moist snuff have the highest level of available nicotine (21).

Using spit tobacco can lead to nicotine dependence or addiction (4,27). The U.S. Surgeon General, the World Health Organization and the American Psychiatric Association have all recognized chronic tobacco use as drug addiction (27). Further, the Surgeon General has compared nicotine to other addictive drugs such as cocaine or heroin. While nicotine is a legal drug and the others are not, the processes that determine nicotine addiction are similar to those that determine addiction to other drugs.[f]

Suggested health problems under study are high cholesterol levels (37,42), accelerated coronary heart disease (28,35), upper digestive tract cancers (35,41), high blood pressure (28,35,43), and cancers of the throat, pancreas, prostate in males (44) and urinary tract (35).

- ***Nicotine addiction plays a role in long-term health consequences.***

Addiction to spit tobacco combined with the serious health effects of long-term tobacco use assure that many young users face an unhealthy future. Researchers agree a relationship between spit tobacco and cigarette use exists, although the dynamics and sequence are not clear (45,46). One researcher explains it this way, "It appears that the use of one nicotine product is associated with the initiation of another nicotine product. The use of [spit tobacco] is far more likely to precede cigarette smoking than the other way around." (46) The majority of key informants say many young people start their tobacco use with spit tobacco. Additionally, they say many also smoke or will switch to cigarettes in the future. The First International Conference on Smokeless Tobacco, in recognizing the addictive nature of nicotine, concluded "...there is a clear potential for [spit tobacco] to serve as a gateway substance for cigarette smoking and the use of other addictive substances, [i.e. alcohol, illicit drugs]." (47)

Therefore, the probable course of events is: 1) nicotine addiction leads to continuous tobacco use, either as spit tobacco, cigarettes or both, which leads to 2) continuous intake of carcinogenic or other harmful substances, that, in time, may lead to 3) cancers and a whole host of other disease processes.

- ***Daily, sustained use is often the pattern for young people; many admit to nicotine addiction.***

Young spit tobacco users follow a frequent and intense use pattern. One national study found that daily use rates among current users were 55 percent for young people between 11 and 15 years old, and 70 percent for those between 16 and 18 years old (48). Further, regional and State data show young male spit tobacco users consume, on average, between one to three or more cans of snuff per week and one to three or more pouches of chewing tobacco (11,13,49). Our 1992 users averaged 3.5 cans of snuff or almost 1 pouch of chewing tobacco a week. They averaged use of over six times per day, with over one-fourth using ten or more times a day -- a nicotine intake equivalent to 1.5 to 2 packs of cigarettes. Young users also keep each tobacco dose in their mouths anywhere from 10 minutes to one hour, with more than twenty minutes not uncommon (17,49-51). Our 1986 and 1992 users typically held their dip or chaw 25 to 30 minutes, with most keeping it in over 30 minutes, and often up to an hour.

Addiction continues to be a problem for young users. The 1986 OIG report on spit tobacco found 37 percent of young users continued spit tobacco use because they were addicted. Many wanted to quit and had tried

> "I've quit 4 or 5 times, but only for a week. Then I think, using hasn't done any damage, so I go back to it."

members of major and minor league professional baseball during spring training of 1988. Thirty-eight percent of the players currently used spit tobacco. Most used moist snuff, Copenhagen and Skoal being the most preferred brands (37). By 1991, current use by baseball players reached 45 percent -- "unprecedented in the history of baseball and the nation."(26)

Product placement in stores and spit tobacco imitations also influence young people. Local studies have found distributors locate moist snuff in stores that youth visit frequently, and they often place it next to cigarettes, candy and snacks (26,56-58). Chewing gum manufacturers have a shredded gum called "Big League Chew" and a tape-shaped bubble gum rolled to fit into a flat, round metal container, similar to a snuff tin (59). Sunflower seeds are marketed as "Dugout Chew" and other products resembling snuff, such as beef jerky, have been marketed to youth. The National Dental Tobacco Free Steering Committee took exception to this marketing technique and has resolved to "condemn the manufacturing and marketing of candy and gum packaged to look like tobacco products." (60)

Extensive And Successful Promotion Of Spit Tobacco Victimizes Susceptible Youth.

- ***Moist snuff consumption has nearly tripled since 1972, making it a very profitable business.***

Moist snuff and looseleaf chewing tobacco are the two most popular forms of spit tobacco, but their growth patterns have been quite different (61). Since 1972, pounds of moist snuff consumed has grown steadily until, in 1991, it was nearly 3 times the 1972 level. Consumption has increased by 24 percent since 1986. On the other hand, pounds of looseleaf tobacco consumed grew until 1980, when it peaked, and then declined 12 percent by 1991. The U.S. Department of Agriculture predicts that moist snuff production in 1992 will increase from the 1991 level (62). As mentioned earlier, in 1970 men over the age of 55 were the heaviest users of snuff; by 1985, males under the age of 19 had replaced them. Seventy-eight percent of our 1992 users prefer moist snuff, particularly once they became regular users.

One company, U.S. Tobacco Company (UST), dominates the spit tobacco industry with its moist snuff products, the most popular being Copenhagen and Skoal (61). In fact, key informants and our 1992 users named Copenhagen and Skoal as top choices among spit tobacco options. In 1991, UST produced 87 percent of the moist snuff consumed and held the largest market share of all spit tobacco produced. In a 1991 Annual Report, UST said net sales for the tobacco segment rose 18 percent to $773.2 million; net earnings reached $265.9 million, a 19 percent increase from the prior year due primarily to the tobacco segment (63). A July 1992 Kidder Advisory on stock market investments characterized UST as "very well positioned in a high-growth, highly profitable industry."

The growth in moist snuff sales is remarkable considering the publicity surrounding the death of Sean Marsee eight years ago (64). Marsee started using Copenhagen at age 13

young user can build up a nicotine tolerance (12,25,26,67). Another example comes from a key informant who, at a spit tobacco sponsored event, saw imprinted hats and jackets in small sizes that would not fit adults. The industry has repeatedly disavowed the charge that they intend to sell their products to minors (69-71). They also remind their critics that the use of tobacco products for people age 18 or older is legal and that manufacturers have a right to promote them at events designed for adult audiences.

Even if the spit tobacco companies are successful in portraying spit tobacco use as an adult behavior, their promotions may still influence many under-age youth to use their products (45). Emulating adult behavior or adopting an image of masculinity, athleticism or toughness are very attractive to young people, especially boys (45). Whether they are the intended consumers or not, large numbers of young people from many backgrounds do obtain and use spit tobacco. Many become nicotine addicts before they finish school, and many are seriously endangering their future health.

Spit Tobacco Laws And Their Enforcement Are Weak And Ineffective.

- ***American public policy on spit tobacco control is inconsistent with other tobacco products.***

Unlike the United States, many other developed countries totally ban spit tobacco sale and use. In 1987 the Study Group on spit tobacco of the World Health Organization recommended that countries with no history of spit tobacco use should ban it (26). By 1990 New Zealand, Australia, Hong Kong, Ireland and Belgium had banned spit tobacco. By 1992 the European Economic Community had given preliminary approval to a ban on these products in its 13 member nations (26). The Secretary of Health and Human Services, has openly criticized American policies that permit the promotion of spit tobacco, a product which is so harmful to good health (72).

American policy for the regulation and taxation of substances intended for adult use only, such as spit tobacco, cigarettes and alcohol, is internally inconsistent. Twenty-one is the national legal age for purchasing alcohol; 18 is the age for tobacco. As late as 1990, 16 States had no excise tax on spit tobacco while all States had excise taxes on cigarettes (73). In 1989 Federal excise tax on cigarettes was 16 cents per pack, but only 1.8 cents on a tin of snuff and 1 cent on a pouch of chewing tobacco (74). The Federal excise tax on spit tobacco was only recently restored in 1985 (26). Finally, all States require retail alcohol vendors to be licensed; only 22 States require the same for tobacco (75).

- ***The Comprehensive Smokeless Tobacco Health Education Act of 1986 has not worked as intended.***

The 1986 Act placed advertising and packaging restrictions on spit tobacco products. Three-fourths of key informants agree the Act has not been an effective deterrent to spit tobacco use by young people. Criticisms from the key informants and some research/policy data are: 1) young people don't read or don't heed the warning labels on

should be a requirement for warning signs at the point of purchase, provision to revoke merchant licenses for violation and a ban on the distribution of free tobacco products (68). Only 22 States prohibit distribution of tobacco product samples to minors (75).

States and localities do not enforce existing tobacco control and access laws. In 1992 the OIG surveyed the States about tobacco control and access, and found virtually none enforce their laws prohibiting the sale of tobacco to minors (81). Eighty-five percent of key informants believe enforcement of spit tobacco sales laws is ineffective. Most favor stronger enforcement including stiffer penalties and license revocations for selling spit tobacco to minors. Smaller, community-based studies show that minors can generally purchase tobacco products at will, and vendors rarely suffer the legal consequences (56,57,77,82). In fact, in studies across the country, it has been shown that, on average, 75 percent of retail stores sell tobacco to minors as young as age 12 (68). Fifty-seven percent of our 1992 spit tobacco users reported it was easy to purchase spit tobacco as a minor. Fifty-three percent stated they were "never" or "hardly ever asked" for proof of age when they purchased their spit tobacco.

- ***Experts favor revisions to spit tobacco control policies.***

Revisions to policies on product promotion and legal restrictions would improve attempts to discourage spit tobacco use by young people. For both areas, key informants strongly support requiring warning labels on billboards and raising the legal age for purchasing to 21. They also see higher Federal and State excise taxes on spit tobacco as beneficial. Research has shown higher prices will effectively discourage purchase by young people (35,74). In fact, other than developing serious health problems from use, higher price was the only strong reason to quit for many of our 1992 users. Finally, key informants support further promotional restrictions: banning giveaway items imprinted with brand logos, or appearance of brand logos in televised events; elimination of advertising messages implying spit tobacco is a healthier choice than cigarettes; and limiting the number or type of events a spit tobacco company can sponsor.

Under-Age Spit Tobacco Use Is A Community Problem, Requiring Broad Interventions.

- ***Spit tobacco use is often socially acceptable within a community.***

Communities sometimes do not understand the social and legal issues of tobacco use until their consciousness is raised. Several local studies have shown that adherence to laws restricting tobacco sales to minors greatly improved after educating the community in general, and the tobacco vendors in particular, on the tobacco access laws (77,82). A community physician suggested local education requires demonstrating how easy it is for youth to obtain tobacco, generating concern over the health problems of tobacco use, and subsequently developing local and school policies on tobacco control (83). All key informants agree parents need to be better informed of the adverse effects of spit tobacco use. Community education may even have to extend to legislators or the judiciary who

but their knowledge was weak for gum recession (24%), throat cancer (20%), addiction (13%) and leukoplakia (7%). Even if young users are intellectually aware of the effects, they don't really believe that spit tobacco endangers their health -- "It can't happen to me" syndrome.

Experts see a need to improve the frequency and content of school curricula addressing spit tobacco. School education about the health consequences of tobacco use was mandated by law in only 20 States in 1989 (68). Two-thirds of key informants recommend the annual inclusion of the health effects of spit tobacco use in school curricula for elementary, junior high and senior high schools. Eighty-two percent say spit tobacco education should start in the third grade or earlier, even in kindergarten; others agree (18,68,85-87). Thus, tailoring curricula to the students' age level is necessary (55,86-89). One analysis of 28 examples of spit tobacco educational materials (1976 to 1986) found the focus was correct (young white males), but social and reading levels tended to be aimed at 7th grade or older (89). Just providing information on the hazards of tobacco use, which many schools do, has been found largely ineffective (68). Simply teaching youth to say "no" does little to combat peer pressure to adopt peer group opinions and values and to gain group acceptance (90,91). So, linking the course content to what motivates young people to use is another improvement (55,68,90,92).

- ***Educated, proactive health professionals are important to community intervention.***

Health professionals have a unique opportunity to discourage spit tobacco use. The dental profession treats 62 percent of the American population within any one year interval, including 75 percent of persons age 5 to 17 (60). Given this large amount of contact, the NCI calls the profession a resource to combat tobacco use by completing better oral examinations, influencing patients' avoidance or discontinuance of tobacco use, and promoting the adoption of comprehensive tobacco control policies (60). In addition, physicians are seen as a credible source of cessation advice. At the time of consultation, patients are sensitized to their health, thus creating the "teachable moment." (68) Key informants unanimously favored strong, proactive education, intervention, counseling and advocacy roles for health professionals.

Many health professionals, however, are not doing as well as they could. Despite the fact that most physicians believe it is their responsibility to encourage their patient to abstain from using tobacco, many fail to do so routinely with all patients (68). Some are ambivalent about the economic value of interventions, and they have a fear of losing patients (93). Key informants strongly believed that health professionals, particularly dentists, dental hygienists, school nurses and family physicians, are not sufficiently trained to prevent or intervene in spit tobacco use. Basic skills needed are the ability to recognize spit tobacco use and its adverse effects, and the signs and effects of addiction. One key informant and his colleagues wrote, "The full extent of tobacco's contributions to oral disease often remains unrecognized by oral health professionals." (33) Counseling techniques, and available educational and cessation materials are also appropriate training topics, either in the professional schools or as part of continuing education afterwards.

CONCLUSIONS

The responsibility for addressing the problem of spit tobacco use by young people rests with many groups at the national, State and local levels. At the Federal level, besides the Department of Health and Human Services (primarily the Public Health Service), several other Departments or agencies are involved -- the Federal Trade Commission, the Department of Agriculture, and the Department of the Treasury. Below, we lay out four broad areas for action by all responsible groups. We strongly encourage the Department of Health and Human Services to take the lead in coordinating efforts to deal with the problem of spit tobacco use. We believe these efforts should have four major goals:

- To convince the general public, including young people, that spit tobacco is a dangerous and addictive drug, not a safe alternative to smoking.

- Based on this awareness, to encourage States and communities to take actions that effectively combat young people's spit tobacco use.

- To solicit support from health care providers and organized athletics which have a unique and important role in deterring spit tobacco use.

- To provide leadership for the re-examination of legislation and regulatory provisions governing spit tobacco and for assuring a coordinated plan for deterring spit tobacco use.

Promote Community Awareness And Action.

Responsible groups should lead a public information campaign to raise community awareness and concern about the problem of youth use of spit tobacco. To support community efforts to curtail use, these groups should actively promote tobacco control policies for schools in cooperation with the Federal and State Education departments.

Support Improved State And Local Tobacco Control.

Responsible groups should support State and local laws that strongly restrict tobacco sales to minors and the sustained enforcement of these laws.

Seek The Support And Involvement Of Health Care Providers And Organized Athletics.

Medical and dental professionals have a unique opportunity to positively impact the problem of spit tobacco use by young people. Responsible groups should make every effort to equip, encourage and involve these professionals. These groups should also actively promote collaborative efforts to deter spit tobacco use by athletes at all levels.

ENDNOTES

a. While current, national data is not available, data from these select States indicate a potentially serious problem with spit tobacco use exists in all States.

b. "Despite varying methodologies among the national surveys, sufficient commonalities permit meaningful comparisons....[Comparisons] between the 1970 NHIS and 1985 CPS for the purpose of examining trends are appropriate." (4)

c. In addition, the 1985 NIDA Household Survey showed past year spit tobacco use by males age 12 to 17 had risen to only one percent behind 18 to 24 year olds, and was more than double the rate of all other adult male age groups (144). In fact, 12-17 year old males had the highest past year use of all male age groups in the Western (25 percent), North Central (22 percent) and Southern (24 percent) regions of the nation.

d. NCAA comparative data from 1985 and 1989 on spit tobacco use. Spit tobacco use increased across both genders, all racial/ethnic groups, all sports, all NCAA divisions and in most geographical regions of the country.

Percent use of spit tobacco in last 12 months by student-athletes by sport					
Male	**1985**	**1989**	**Female**	**1985**	**1989**
Baseball	45%	57%	Softball	8%	9%
Basketball	8%	15%	Basketball	2%	4%
Football	30%	40%	Swimming	3%	3%
Track	9%	20%	Track	3%	5%
Tennis	12%	29%	Tennis	-	-
Percent use by student-athletes by region and college division					
East	15%	25%	**Division I**	19%	27%
Midwest	19%	33%	**Division II**	23%	29%
South	25%	23%	**Division III**	19%	27%
West	24%	24%	Source: NCAA (5), 1989 '85 (N = 2,039), '89 (N = 2,282)		

e. N = 3,159 in 1986 and N = 2,852 in 1989. Unfortunately, spit tobacco questions were omitted from the Senior Survey in 1990 and 1991.

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APPENDIX A

PROFILES OF 1992 SPIT TOBACCO USERS

These are brief profiles of some young users we interviewed for the study.

1. **Jack** from Arkansas, 17 years old, white.

   Jack has used spit tobacco regularly since the sixth grade. His father was a professional bass fisherman who had received free spit tobacco samples. His father died of oral cancer after using for 40 years. Jack has health problems from using -- receding, sore gums, peeling skin inside his cheek, sores on his tongue and frequent sore throats. In fact, he admits he is addicted, but he hasn't tried to quit. His special interest is high school rodeo; using spit tobacco is a "thing all real cowboys should do." He says his school coaches borrow spit tobacco from him "all the time."

2. **Carlos** from Texas, 16 years old, Hispanic.

   Carlos has been a regular user since the ninth grade. Baseball is his sport -- he plays on his school team, and he watches it on television. He started using spit tobacco because the older baseball players used; it was just something everyone else did. Now he's hooked and says he can't play ball without it due to cravings. Carlos has tried to quit three times with no success. He thinks spit tobacco probably isn't good for him, but he and his parents think it's better than smoking or taking drugs.

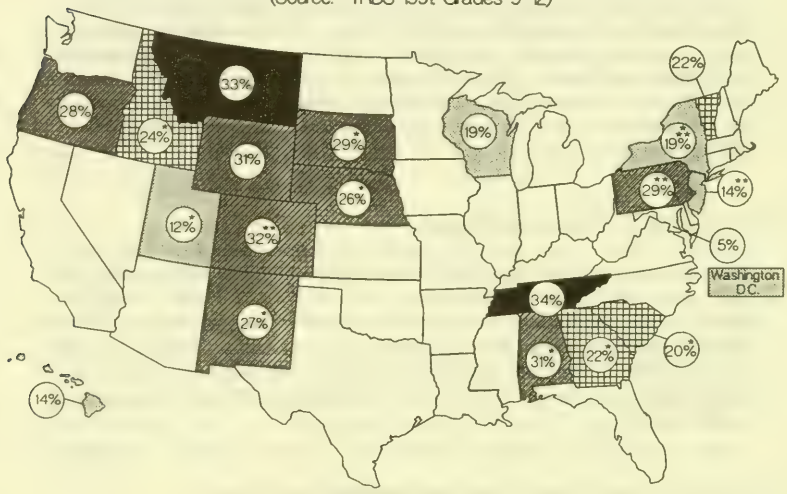
3. **Cal, 7, Lee, 10, and Scooter, 11** are three brothers from Florida

   All three boys started using spit tobacco very young -- Cal has used chewing tobacco regularly since Kindergarten, Lee since he was 3 years old and Scooter since second grade. Cal and Lee say they now use all day long and sometimes go to sleep with spit tobacco in their mouths. Their father, who buys spit tobacco for them, has used chewing tobacco for over 25 years and says he has no bad health effects. The boys do agree, though, that regular use can cause mouth or throat cancer. The town they live in has a drug problem. Dad feels that spit tobacco is a better alternative to hard drugs ...and candy.

APPENDIX B

Males Currently[+] Use Spit Tobacco At High Rates In Twenty States.[1]

(Source: YRBS 1991 Grades 9–12)



19% or less | 20–24% (over 1 in 5 males) | 25–32% (over 1 in 4 males) | 33% or more (over 1 in 3 males)

* Weighted data ** Surveys did not include students from the largest city. + Preceding 30 days

[1] Includes Washington, D.C.

APPENDIX C

SUMMARY OF NATIONAL AND REGIONAL PREVALENCE STUDIES

Regional Studies - Spit Tobacco

Bib	Investigators/Location	Age/Grade	Sample N	Miscellaneous Study Information	Current Use (Last 30 days) of Spit Tobacco [1] Used Regularly	
					Males	Females
13	Schlife (1987) 8 rural regions in Alaska	K-12th grades	4,965	Native American Study	33.7% [1]	27.5% [1]
	Schinke (1986) 3 reservations & 1 urban area, WA	Mean Age = 13.8	254	Native American Study	20.2% [1]	27.4% [1]
	Schinke (1987) Washington, Alaska	Mean Age = 12.3	144	Native American Study	42.6% [1]	34.0% [1]
	Wolfe and Carlos (1987) Navajo Boarding School, NM	9-10th grades	226	Native American Study	55.9% [1]	
	Jewett (1988) Rosebud Reservation, SD	K-6th grades 7-12th grades	1,010 571	Native Amercian Study	39.2% [1]	35.1% [1]
	Hall and Dexter (1989) 3 tribes, 15 schools, WA	6,9,11th grades	257	Native American Study	34.0% [1]	24.0% [1]
	Jewett (1988) 4 reservations & urban non-Indians, South Dakota and Montana	7-12th grades	1056	Native American Study	36.2% [1]	32.4% [1]
	Batliner (1988) South Dakota & Montana	4-8th grades	623	Native American Study	47.0% [1]	45.0% [1]
	Foster (1988) Boarding School, SD	7-12th grades	114	Native American Study	38.0% [1]	37.0% [1]
13	Foster (1988) South Dakota	K-12th grades	195	Non-Native Americans (Comparative group to Native Americans)	18.4% [1]	8.7% [1]
	Hall & Dexter (1988) Washington	6,8,11th grades	853	Non-Native Americans (Comparative group to Native Americans)	20.0% [1]	4.0% [1]
	Jewett (1988) Metropolitan Minneapolis	7-12th grades	23,693	Non-Native Americans (Comparative group to Native Americans)	7.8% [1]	0.0% [1]
	Jewett (1988) Greater Minneapolis	7-12th grades	12,590	Non-Native Americans (Comparative group to Native Americans)	6.8% [1]	0.0% [1]

Bib	Investigators/Location	Age/Grade	Sample N	Miscellaneous Study Information	Current Use (Last 30 days) of Spit Tobacco [1] Used Regularly	
					Males	Females
13	Bathiner (1988) South Dakota, Montana & Nebraska	4-8th grades	527	Non-Native Americans (Comparative group to Native Americans)	14.6% [1]	1.6% [1]
52	Marty, et al. (1986) Rural community, NW Arkansas	10-12th grades Mean age = 15.9	179	Predominantly Caucasian, blue collar, rural population	31.8%	2.3%
19a	University of Kentucky 1 Metro. & 1 non-Metro. county	7-12th grades	1,067	Phase 1: Saliva sample collected (471 sent for biochemical testing) Phase 2: 85 metro 8th graders were exposed to 3-sessions of education intervention.	Use in Past 6 Days: 42% of those in households involved in tobacco	
19b	West Virginia University WV Public Schools	5-12th grades	4,230	Stratified random sample	17% 5-6th 29% 7-9th 39% 10-12th	3% 5-6th 1% 7-9th 1% 10-12th
12	Brownson, et al. (1990) 78 Missouri Schools	5,8,12th grades	5,431	Representative sample, rural counties were oversampled	Use in Previous 7 Days Urban: / Rural: 5th 1% / 5% 8th 13% / 17% 12th 15% / 31%	
51	Jones & Pyper (1986) Dane County, Wisconsin	7-12th grades	2,181	Stratified random sample	Male Only: > 1/wk: / Daily Use: 7th 9% / 3% 8th 12% / 6% 9th 12% / 3% 10th 16% / 8% 11th 14% / 11% 12th 22% / 15%	
102	Kegeles, et. al. (1989) 59 Connecticut towns & 106 schools	7-12th grades	7,457		Daily Use: 7th 2.0% 8th 5.3% 9th 7.1% 10th 5.5% 11th 7.1% 12th 8.0%	Daily Use: 7th 4% 8th 4% 9th 5% 10th 0% 11th 3% 12th 2%

Bib	Investigators/Location	Age/Grade	Sample N	Miscellaneous Study Information	Current Use (Last 30 days) of Spit Tobacco [1] Used Regularly	
					Males	Females
18	Leopardi, et. al. (1989) Davis County, Utah All 11 Junior highs & 7 Senior highs	7-12th grades	8,902	Survey I - 16-item prevalence survey Survey II - 473 males in sports classes had survey & oral exam.	Weekly Use: 12.1% Total 10.2% JH 15.0% SH	Weekly Use: 1.1% Total 1.1% JH 1.0% SH
14	North Dakota State Department of Health and Consolidated Laboratories (1991), 212 ND School Districts	7-12th grades	36,693		7-12th 20.4% Sioux county 62.9%	
20	State Department of Education (89-90) Oklahoma	6,8,10,12th grades		1989-90 Drug & Alcohol Use Among Oklahoma Students.	Daily Use: 6th 3% 8th 6% 10th 8% 12th 11%	
103	Bauman, et. al. (1989) 10 SMSA's, Southeastern U.S.	12-14 yrs	21,203	Probability samples of households.	Ages White: 0-4 .2% 5-9 .3% 10-14 3.5% 15-19 11.4% 20-24 10.6%	Ages White: 0-4 .1% 5-9 .0% 10-14 .1% 15-19 .2% 20-24 .5%
104	Peterson, Marek, & Mann (1989) Schools in Washington state.	10th grade	1,631	14 rural and suburban school districts.	10th Graders Last Month 25.3% Last Week 17.7%	10th Graders Last Month 2.8% Last Week 1.4%
8	Tri-Agency Tobacco Free Project (1991), Maine	5,7,9,12th grade	26,911	Maine Youth Tobacco Use Survey	Last Week 5th 1.4% 7th 2.7% 9th 6.6% 12th 10.2%	
9	Tri-Agency Tobacco Free Project (1989), Maine	5,7,9 & 12th grade	22,147	Maine Youth Tobacco Use Survey	Last Week 5th 1.5% 7th 4.0% 9th 6.0% 12th 8.7%	

Bib	Investigators/Location	Age/Grade	Sample N	Miscellaneous Study Information	Current Use (Last 30 days) of Spit Tobacco [1] Used Regularly	
					Males	Females
10	Tri-Agency Tobacco Free Project (1987), Maine	5,7,9 & 12th grade	28,000	Maine Youth Tobacco Use Survey	Last Week 5th 1.8% 7th 3.5% 9th 7.8% 12th 8.3%	
49	Simon, Stotts & Pollard (1992) West Virginia	5th - 12th grade	4,230	Stratified random sample by student achievement & socialeconomic status. Includes no racial and ethnic categories.	Almost Daily Use 6th 6.3% 7th 9.6% 8th 11.0% 9th 22.6% 10th 19.5% 11th 21.9% 12th 20.8%	
11	Illinois Department of Public Health (1987), Illinois (excluding Chicago)	5,7,9, & 11th grades 9-19 years	7,118	Random sampling of 65 schools in Illinois.	Metro. Non-Metro. 5th 3.7% 4.8% 7th 7.2% 11.8% 9th 14.6% 26.7% 11th 16.4% 28.4%	
85	Center for Health Statistics & Bureau of Community Health & Prevention, (1988), Wisconsin	18-24 years	2,400	Randomly selected adult households.	7%	
85a	Murray, D., (1986) Wisconsin Public Schools	9th grade	3,500 +	Randomly selected 9th graders in public schools.	Last Week 5.0%	Previous Day 3.0%
105	University of Minnesota 50 different Native American tribes	7-12th grades	14,000	Adolescent Health Survey of Native American adolescents.	Daily 7th 10.7% 8th 11.5% 9th 14.9% 10th 16.0% 11th 18.8% 12th 20.3%	Daily 7th 6.4% 8th 7.1% 9th 10.4% 10th 8.9% 11th 7.9% 12th 6.4%
106	Pomrehn, et. al. Three communities in Eastern Iowa	10,11 & 12th grades	1245	Adolescent Tobacco Use Prevention Study	10th 12.5% 11th 15.9% 12th 13.6%	

Bib	Investigators/Location	Age/Grade	Sample N	Miscellaneous Study Information	Current Use (Last 30 days) of Spit Tobacco [1] Used Regularly	
					Males	Females
107	Edumson, et. al. (1987) East Carolina University	College Freshman-Senior	289	Random sample of undergraduate male students at East Carolina University. Looked at personality characteristics.	Dipping 17-18 yrs 18.3% 19-20 yrs 14.8% Chewing 17-18 yrs 12.2% 19-20 yrs 10.2%	
7	Connecticut Department of Health Services, (89-90)	4th-12th grades	6981 (7-12th grades)	Connecticut Health Check	4th 1.0% 5th 1.0% 7th 4.0% 8th 1.0% 9th 3.0% 10th 7.0% 11th 7.0% 12th 10.0%	
108	Youth Risk Behavior Survey (1991) Nebraska	9-12th grades	2450	School based survey (YRBS) Weighted data	Total 26.1% ≤ 15 19.6% 16,17 26.6% ≥ 18 36.1% White 27.8% Black 3.0% Hispanic 18.5% Other 24.2%	Total 2.4% ≤ 15 2.7% 16,17 2.5% ≥ 18 1.6%
109	Youth Risk Behavior Survey (1991) Alabama	9-12th grades	2455	School based survey (YRBS) Weighted data	Total 30.7% ≤ 15 31.3% 16,17 31.0% ≥ 18 28.6% White 39.2% Black 6.8% Hispanic ---- Other 16.9%	Total 2.0%

Bib	Investigators/Location	Age/Grade	Sample N	Miscellaneous Study Information	Current Use (Last 30 days) of Spit Tobacco [1] Used Regularly	
					Males	Females
110	Youth Risk Behavior Survey (1990) Tennessee	9-12th grades	1889	School based survey (YRBS) Unweighted data	≤ 15 20.2% 16,17 24.3% ≥ 18 27.5% White 28.6% Black 2.8% Hispanic ---- Other ----	≤ 15 1.7% 16,17 1.4% ≥ 18 .9%
111	Youth Risk Behavior Survey (1991) Tennessee	9-12th grades	2457	School based survey (YRBS) Unweighted data	Total 34.4% ≤ 15 30.6% 16,17 35.8% ≥ 18 36.6% White 35.8% Black 22.1% Other 29.0%	Total 1.2% ≤ 15 .7% 16,17 1.5% ≥ 18 1.6%
112	Youth Risk Behavior Survey (1991) Pennsylvania	9-12th grades	2192	School based survey (YRBS) Unweighted data	Total 29.0% ≤ 15 28.0% 16,17 29.0% ≥ 18 30.8% White 30.4% Black 17.3% Hispanic 5.9% Other 30.0%	Total 1.8% ≤ 15 1.0% 16,17 2.3% ≥ 18 1.6% White 1.7% Black --- Hispanic --- Other 3.1%
113	Youth Risk Behavior Survey (1990) North Carolina	9-12th grades		School based survey (YRBS)	9th, White 23.0% 12th,White 18.0% 9th,Black 7.0% 12th,Black 2.0 %	
114	Youth Risk Behavior Survey (1990) Mississippi	9-12th grades		School based survey (YRBS)	19%	3.0%
3	Youth Risk Behavior Survey (1991) Georgia	9-12th grades		School based survey (YRBS) Weighted data	22%	2.0%

Bib	Investigators/Location	Age/Grade	Sample N	Miscellaneous Study Information	Current Use (Last 30 days) of Spit Tobacco [1] Used Regularly	
					Males	Females
3	Youth Risk Behavior Survey (1991) Idaho	9-12th grades		School based survey (YRBS) Weighted data	24%	3.0%
3	Youth Risk Behavior Survey (1991) New Mexico	9-12th grades		School based survey (YRBS) Weighted data	27%	4.0%
3	Youth Risk Behavior Survey (1991) New York	9-12th grades		School based survey (YRBS) Weighted data excludes students from the largest city.	19%	2.0%
3	Youth Risk Behavior Survey (1991) South Carolina	9-12th grades		School based survey (YRBS) Weighted data	20%	2.0%
3	Youth Risk Behavior Survey (1991) South Dakota	9-12th grades		School based survey (YRBS) Weighted data	29%	10.0%
3	Youth Risk Behavior Survey (1991) Utah	9-12th grades		School based survey (YRBS) Weighted data	12%	2.0%
3	Youth Risk Behavior Survey (1991) Colorado	9-12th grades		School based survey (YRBS) Unweighted data	32%	6.0%
3	Youth Risk Behavior Survey (1991) Washington D.C.	9-12th grades		School based survey (YRBS) Unweighted data	5.0%	2.0%
3	Youth Risk Behavior Survey (1991) Hawaii	9-12th grades		School based survey (YRBS) Unweighted data	14%	2.0%
3	Youth Risk Behavior Survey (1991) Montana	9-12th grades		School based survey (YRBS) Unweighted data	33%	7.0%
3	Youth Risk Behavior Survey (1991) New Hampshire	9-12th grades		School based survey (YRBS) Unweighted data	22%	4.0%
3	Youth Risk Behavior Survey (1991) New Jersey	9-12th grades		School based survey (YRBS) Unweighted data excludes students from the largest city.	14%	2.0%
3	Youth Risk Behavior Survey (1991) Oregon	9-12th grades		School based survey (YRBS) Unweighted data	28%	5.0%

Bib	Investigators/Location	Age/Grade	Sample N	Miscellaneous Study Information	Current Use (Last 30 days) of Spit Tobacco [1] Used Regularly	
					Males	Females
3	Youth Risk Behavior Survey (1991) Pennsylvania	9-12th grades		School based survey (YRBS) Unweighted data excludes students from the largest city.	29%	2.0%
3	Youth Risk Behavior Survey (1991) Wisconsin	9-12th grades		School based survey (YRBS) Unweighted data	19%	3.0%
3	Youth Risk Behavior Survey (1991) Wyoming	9-12th grades		School based survey (YRBS) Unweighted data	31%	5.0%
68	Youth Risk Behavior Survey (1991-92)	9-12th grades		School based survey (YRBS)	White NA 9-12th 29% 39% 9th 24% 38% 10th 31% 34% 11th 30% 47% 12th 32% 35%	White NA 9-12th 4% 13% 9th 3% 11% 10th 5% 13% 11th 4% 16% 12th 4% 14%

National Studies - Spit Tobacco

Bib	Investigators/Location	Age/Grade	Sample N	Miscellaneous Study Information	Current Use (Last 30 days) of Spit Tobacco [1] Used Regularly	
					Males	Females
97	U.S. Department of Health and Human Services (1991) High School students in U.S.	9-12th grades	11,631	National School Based Youth Risk Behavior Survey (YRBS) 1990 Data	Total 19.1% White 23.9% Black 3.1% Hispanic 10.9% Seniors 21.1% Sr./White 26.0%	Total 1.4% White 1.5% Black .8% Hispanic 1.0%
1	U.S. Department of Health and Human Services (1992) High School students in U.S.	9-12th grades	12,272	National School Based Youth Risk Behavior Survey (YRBS) 1991 Data	Total 19.2% White 23.6% Black 3.6% Hispanic 10.7%	Total 1.3% White 1.4% Black .7% Hispanic .6%
98	Glover, Laflin, & Edwards (1989) College students across eight regions	Mean age=20.8 yrs 1/3 were freshman	5,894	Stratified, random, multi-cluster sample of physical activity classes among 72 colleges and universities throughout the U.S. 1986 Data	12% of all college age	
99	National Institute on Drug Abuse (1988)	Ages 12-17 & 18-25		NIDA Household Survey on drug abuse 1988 Data	Ages 12-17 6.6% Ages 18-25 12.3%	Ages 12-17 ---- Ages 18-25 ----
100	National Institute on Drug Abuse (1990)	Ages 12-17 & 18-25		NIDA Household Survey on drug abuse 1990 Data	Ages 12-17 7.4% Ages 18-25 12.1%	Ages 12-17 ---- Ages 18-25 ----
101	National Institute on Drug Abuse (1991)	Ages 12-17 & 18-25		NIDA Household Survey on drug abuse 1991 Data	Ages 12-17 5.3% Ages 18-25 11.6%	Ages 12-17 .5% Ages 18-25 .4%
6	National Institute on Drug Abuse (1986)	12th grade	3159	NIDA Senior Survey 1986 Data	Seniors 22.2% Sr./White 25.7%	Seniors 1.6%
6	National Institute on Drug Abuse (1987)	12th grade	3357	NIDA Senior Survey 1987 Data	Seniors 22.8% Sr./White 25.4%	Seniors .6%
6	National Institute on Drug Abuse (1988)	12th grade	3378	NIDA Senior Survey 1988 Data	Seniors 19.9% Sr./White 21.9%	Seniors 1.7%
6	National Institute on Drug Abuse (1989)	12th grade	2852	NIDA Senior Survey 1989 Data	Seniors 16% Sr./White 19%	Seniors 1.2%

APPENDIX D

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STATEMENT OF

P. GERALD THACKER

ACTING COMMISSIONER

PUBLIC BUILDINGS SERVICE

GENERAL SERVICES ADMINISTRATION

BEFORE THE

SUBCOMMITTEE ON PUBLIC BUILDINGS AND GROUNDS

COMMITTEE ON PUBLIC WORKS AND TRANSPORTATION

U.S. HOUSE OF REPRESENTATIVES

MARCH 11, 1993

GOOD MORNING MR. CHAIRMAN, AND MEMBERS OF THE SUBCOMMITTEE. THANK YOU FOR THE OPPORTUNITY TO APPEAR BEFORE YOU TODAY TO DISCUSS H.R. 881, A BILL TO PROHIBIT SMOKING IN FEDERAL BUILDINGS.

WHEN TESTIFYING BEFORE THIS SUBCOMMITTEE ON FEBRUARY 23RD, I NOTED THAT THE GENERAL SERVICES ADMINISTRATION'S PUBLIC BUILDINGS SERVICE PROVIDES WORKSPACE FOR MORE THAN 1 MILLION FEDERAL EMPLOYEES. IN ADDITION, NUMEROUS MEMBERS OF THE PUBLIC, SUCH AS SOCIAL SECURITY APPLICANTS, JURORS AND CONTRACTORS VISIT OUR FACILITIES. MANY NON-FEDERAL ACTIVITIES ALSO OCCUPY OUR BUILDINGS. FOR EXAMPLE, THE OLD POST OFFICE IN WASHINGTON, DC, IS A NOTED EXAMPLE OF A MULTI-USE FEDERAL FACILITY THAT SERVES FEDERAL OCCUPANTS AS WELL AS THE SURROUNDING COMMUNITY.

THE PUBLIC BUILDINGS SERVICE IS COMMITTED TO PROVIDING A SAFE AND HEALTHFUL WORK ENVIRONMENT FOR ALL FEDERAL EMPLOYEES AND VISITORS TO OUR BUILDINGS -- WE VIEW THIS AS ONE OF OUR MOST IMPORTANT RESPONSIBILITIES. WITH THE ENVIRONMENTAL PROTECTION AGENCY'S CLASSIFICATION OF ENVIRONMENTAL TOBACCO SMOKE AS A CLASS A CARCINOGEN -- LIKE ASBESTOS AND RADON -- THE SERIOUSNESS OF THE SMOKING ISSUE HAS INCREASED SIGNIFICANTLY.

AS I TESTIFIED IN FEBRUARY, THE CURRENT REGULATION GOVERNING SMOKING IN THE PUBLIC BUILDINGS SERVICE'S FACILITIES DOES NOT ADEQUATELY PROTECT NON-SMOKERS FROM SECOND-HAND SMOKE WHICH IS RECIRCULATED THROUGHOUT BUILDINGS THROUGH THE VENTILATION SYSTEM. WE ARE IN THE PROCESS OF EXAMINING ALTERNATIVES FOR REVISING THE CURRENT REGULATION TO ADDRESS THIS ISSUE. BECAUSE THE PUBLIC BUILDINGS SERVICE IS A PROPERTY MANAGER SERVING ITS CLIENTS, WE CANNOT ACT UNILATERALLY AND MUST CONSIDER THE NEEDS AND VIEWS OF OUR TENANTS IN FORMULATING NEW SMOKING GUIDELINES FOR THE SPACE WE PROVIDE TO THEM.

HOWEVER, AS A PROPERTY MANAGER COMMITTED TO PROVIDING A SAFE AND HEALTHY WORKING ENVIRONMENT FOR ITS TENANTS, THE PUBLIC BUILDINGS SERVICE SUPPORTS THE OBJECTIVE OF H.R. 881, TO PROTECT EMPLOYEES FROM EXPOSURE TO SECONDARY SMOKE IN FEDERAL FACILITIES UNDER THE CONTROL OF THE PUBLIC BUILDINGS SERVICE. IT IS CLEARLY RESPONSIVE TO THE HAZARDS POSED BY ENVIRONMENTAL TOBACCO SMOKE TO NON-SMOKERS AS IDENTIFIED IN THE ENVIRONMENTAL PROTECTION AGENCY'S REPORT. THE PUBLIC BUILDINGS SERVICE BELIEVES A BAN ON INDOOR SMOKING MAY BE THE MOST COST EFFECTIVE WAY TO PROTECT NON-SMOKERS FROM THIS NEWLY CLASSIFIED CLASS A CARCINOGEN. AS I NOTED IN FEBRUARY, THE COST OF ALTERING EXISTING SPACE TO PROVIDE SEPARATELY VENTILATED AREAS WOULD BE SIGNIFICANT -- ABOUT $30 TO $50 PER SQUARE FOOT.

WE DO BELIEVE THERE ARE SOME LOCATIONS, SUCH AS LIVING QUARTERS, OR IN CERTAIN MEDICAL FACILITIES (E.G., EXCEPTIONS WHICH CURRENTLY EXIST AT LOCAL DISCRETION, WITHIN VETERANS' ADMINISTRATION ACUTE CARE FACILITIES), WHERE SMOKING MAY BE PERMITTED PROVIDED NON-SMOKERS ARE NOT UNWILLINGLY EXPOSED. THERE MAY, IN ADDITION, BE OTHER LIMITED AREAS IN WHICH EXCEPTIONS ARE APPROPRIATE.

THERE ARE SPECIFIC PORTIONS OF THE PROPOSED BILL, HOWEVER, WHICH WE BELIEVE SHOULD BE MODIFIED. SECTION 3(B) WOULD REQUIRE THE ADMINISTRATOR OF GENERAL SERVICES TO ISSUE REGULATIONS AND TO TAKE SUCH ACTIONS THAT MAY BE NECESSARY TO INSTITUTE AND ENFORCE THE PROHIBITION ON SMOKING WITHIN THE EXECUTIVE BRANCH. WHILE WE APPRECIATE THE CONFIDENCE AND TRUST IN THE GENERAL SERVICES ADMINISTRATION IMPLIED IN THIS PROVISION, WE BELIEVE IT WOULD BE MORE APPROPRIATE FOR SUCH AUTHORITIES TO BE PLACED ELSEWHERE. AS I NOTED IN FEBRUARY, THE PUBLIC BUILDINGS SERVICE CONTROLS ONLY 10 PERCENT OF THE FEDERAL INVENTORY OF SPACE. MANY OTHER PROPERTY HOLDING DEPARTMENTS AND AGENCIES WOULD BE AFFECTED BY THE PROPOSED LEGISLATION. IN ADDITION, THE GENERAL SERVICES ADMINISTRATION IS NOT AN ENFORCEMENT AGENCY. SECTION 19 OF THE OCCUPATIONAL SAFETY AND HEALTH ACT (OSH ACT) AND EXECUTIVE ORDER 12196 REQUIRE EACH EXECUTIVE AGENCY HEAD TO ENSURE ITS OWN FEDERAL WORKER'S SAFETY AND HEALTH. THEREFORE, WE BELIEVE SECTION 3(B) SHOULD BE MODIFIED TO REQUIRE ALL REAL PROPERTY

HOLDING AGENCIES TO ISSUE REGULATIONS BANNING INDOOR SMOKING WITHIN THE FEDERAL FACILITIES UNDER THEIR OWN JURISDICTION.

WE ALSO NOTE THAT SECTION 3(C) OVERLAPS SECTION 3(B) SINCE MOST COURTS ARE LOCATED IN THE GENERAL SERVICES ADMINISTRATION'S FACILITIES. THIS SHOULD BE ADDRESSED AND THE AUTHORITIES AND RESPONSIBILITIES OF THE RESPECTIVE BRANCHES OF GOVERNMENT SHOULD BE CLARIFIED WHEN THERE IS A MIXED OCCUPANCY.

IN ADDITION, SINCE SMOKING IS AN EMPLOYEE/EMPLOYER WORKPLACE ISSUE, WE BELIEVE IT WOULD BE COUNTERPRODUCTIVE TO REQUIRE ANY AGENCY TO POLICE THE BEHAVIOR OF EMPLOYEES OF ANOTHER AGENCY. AS STATED PREVIOUSLY, UNDER THE OSH ACT AND EXECUTIVE ORDER 12196, AGENCY HEADS HAVE OVERALL OCCUPATIONAL SAFETY AND HEALTH RESPONSIBILITY FOR THEIR EMPLOYEES. THEREFORE, WE RECOMMEND THAT EACH AGENCY BE HELD RESPONSIBLE FOR THE ENFORCEMENT OF THE PROHIBITION GOVERNING THE SPACE THEY OCCUPY. THE DESIGNATION OF A SINGLE AGENCY FOR SUCH PURPOSES SHOULD BE DELETED. WHEN AND WHERE ABUSES MIGHT OCCUR, THEY COULD BE ADDRESSED WITHIN THE EXISTING ADMINISTRATIVE PROCEDURES OF EACH AGENCY TO ENSURE COMPLIANCE. WE BELIEVE THIS IS THE MOST PRACTICAL AND EFFECTIVE WAY OF ENSURING FULL, RAPID, AND COMPLETE COMPLIANCE.

WITH RESPECT TO THE CONSOLIDATED REPORT TO THE CONGRESS AS REQUIRED BY SECTION 4, WE QUESTION ITS NECESSITY AND RECOMMEND THAT CONSIDERATION BE GIVEN TO DELETING THAT REQUIREMENT.

ALSO, WE BELIEVE THE PHRASE "AN ASSESSMENT OF THE LEGAL STATUS OF SMOKING IN PUBLIC PLACES" IN SECTION 4 IS VAGUE, AND NEEDS TO REPHRASED OR DEFINED. IT IS NOT CLEAR IF IT WOULD APPLY ONLY TO FEDERAL PUBLIC PLACES OR ALL PUBLIC PLACES INCLUDING STATE AND LOCAL FACILITIES. IF THE LATTER, THE TIME AND RESOURCES TO COMPILE SUCH A REPORT WOULD BE SIGNIFICANT.

IN CONCLUSION, THE GENERAL SERVICES ADMINISTRATION BELIEVES THAT A BAN SUCH AS THAT PROVIDED FOR H.R. IN 881, WITH MODIFICATIONS, WOULD PROVIDE CLEAR AND DEFINITIVE POLICY GUIDANCE FOR THE FEDERAL WORKPLACE. WHILE WE DEFER TO THE LEGISLATIVE AND JUDICIAL BRANCHES WITH RESPECT TO THOSE PORTIONS OF THE BILL THAT APPLY TO THEM, WE GENERALLY SUPPORT THE PROVISIONS THAT WOULD APPLY TO THE EXECUTIVE BRANCH.

MR. CHAIRMAN, AGAIN, THANK YOU FOR THE OPPORTUNITY TO TESTIFY ON H.R. 881. WE APPRECIATE YOUR CONTINUING INTEREST IN THE PUBLIC BUILDINGS SERVICE. I WOULD BE PLEASED TO RESPOND TO ANY QUESTIONS YOU MAY HAVE.

Good morning ladies and gentlemen. My name is Simon Turner. I'm a Director of the company, Healthy Buildings International. We're a company with over 12 year's experience in studying the internal environment of major office buildings. We have investigated over 1,000 major buildings throughout the world, including many government agencies such as the Department of Health and Human Services, Federal Reserve Banking System, Longworth Congressional Office Building, Supreme Court and the Library of Congress among others.

Our particular area of investigation is the study of indoor pollution, often referred to as sick building syndrome (SBS). This is very much a worldwide problem. The World Health Organization (WHO) has estimated that 30 percent of all new and remodelled buildings suffer from indoor pollution problems. The US EPA points out that the costs to the economy from indoor pollution in this country reach over 12 billion dollars a year in lost productivity alone. You must also add to the lost productivity the massive losses due to absenteeism.

The National Center for Health Statistics has shown that 50 percent of all office absenteeism is due to upper respiratory complaints. In the case of the United States, that loss accounts for 150 million work days lost every year. The costs associated with these losses exceed 15 billion dollars in medical care costs. Indeed, we can also point out that in a study conducted by the Walter Reed Army Institute over a four year period in which they monitored 400,000 people, half the people (200,000) were housed in modern, energy efficient sealed buildings. The other 50 percent were in older, more naturally ventilated buildings. And note, on average they recorded a 50 percent increase in sickness, complaints, and absenteeism from occupants in the sealed, energy efficient buildings. The cause of this is now known to be indoor pollution. When we talk indoor pollution, we're referring to literally hundreds of different chemicals, gases and vapors, many different types of fibers, dusts, and all the microbes — bacteria, fungi, viruses, etc.

It's quite remarkable that in our studies over the last twelve years of 953 major buildings, we've identified what the major pollutants have been in buildings (Table 1). By far the largest source of problems have been due to fungi in about 33 percent of the buildings. Next, and in order of magnitude, we have problems with dusts, followed by low relative humidity, bacteria, formaldehyde, fibrous glass, motor vehicle exhaust fumes, volatile organic compounds, tobacco smoke, high relative humidity and ozone.

It's rather interesting, we've here talking about tobacco today and less than three percent of the problems we have investigated in major buildings have been due to tobacco smoke with a much larger percentage of buildings suffering with things such as fungi, dusts, bacteria, etc. The fact is that all these pollutants in buildings are accumulating for one of three reasons, either poor ventilation, poor filtration or lack of hygiene. It's remarkable to note that nearly 20 percent of all the buildings we've

investigated have been operating with 100 percent recycled air, no outside air whatsoever, practiced in the interests of energy conservation (Table 2).

If you picture yourselves in buildings where you're constantly recycling the same air, passing that air through inefficient filters and moving it through a dirty, contaminated ductwork system, it's only a matter of time before more and more people get sick.

Having said that, we're here today mainly because of the increased focus there is on environmental tobacco smoke as a result of the statement of the EPA that, in their opinion, environmental tobacco smoke is a class A carcinogen.

Let's assume that it were such a carcinogen. First, recognize that the EPA has no statutory authority regulating indoor air quality in the workplace. That is passed to other agencies. Each of these other agencies has lots of experience in dealing with carcinogens in any workplace. For example, OSHA, when they manage risks, including class A carcinogens use Permissible Exposure Limits. These are typically 8 hour time weighted averages, below which they see no cause for concern. In addition, NIOSH have Recommended Exposure Limits for class A carcinogens, below which they see no problem. And then there's the American Conference of Governmental Industrial Hygienists. They use Threshold Limit Values for carcinogens and they define TLVs as concentrations of substances that nearly all workers may be exposed to, day after day, without any adverse health effects.

So let's now look specifically at a common class A carcinogen — benzene (Table 3). You'll notice that the OSHA standard, the permissible exposure limit is 1 ppm and we have a different standard by the industrial hygienists, which is ten times larger at 10 ppm. It seems strange to me that we should have such a discrepancy in the base standard. But then, when we look at the actual measurements in homes. And I'm using here the studies that were published in the Samet and Spengler book Indoor Air Pollution — A Health Perspective (The Johns Hopkins University Press 1991). They — anti-smokers — drew attention to the fact that there was more benzene in the smoking home than the non-smoking home and I accept that fact. But note, we're talking about a differential of 0.0012 ppm, about 1,000 times below the most stringent standard adopted in this country. And far more relevant is the fact that in our study of over 225 offices (we've looked at that number of non-smoking offices and the same number of smoking offices), the difference between the two is only 0.0005 ppm. Most interesting of all, is there is more benzene in the non-smoking home than there is in the smoking office, and our smoking offices include smoking lounges.

The fact is that benzene is a common carcinogen. In the home it is present in all sorts of waxes, polishes, sprays and cleaning compounds. It is present in adhesives and in glues used for hobbies, etc. The significant difference is that homes have no

dedicated ventilating system, when even poorly ventilated offices still have mechanical capability and that's why you're finding more of this carcinogen in the home than in the offices, even the smoking offices.

This is not just work done by ourselves. Holcombe has investigated every single published study of benzene. He found that the average readings in smoking and non-smoking offices in the US fall very much in line with the figures I've quoted you from mine. There is a marginal increase in the smoking office over the non-smoking office, but the very important thing is that there is more benzene in the homes than in the offices and there's even more benzene in vehicle transportation.

We can consider one more class A carcinogen — asbestos (Table 4). OSHA has a permissible exposure limit of 0.2 fibers per cc of air. What does this mean? If we consider a 100,000 square foot building — this would be a typical 10 story building — we can look at what the volume of that building might be and translate it into cc's (cubic centimeters). We have over 28 million cubic centimeters in that building. At 0.2 fibers per cc, the Permissible Exposure Limit is 5.6 million fibers. Thus you could point out that at 5 million fibers of airborne asbestos, that building would be judged as acceptable. Almost ironically, let a few molecules of environmental tobacco smoke in the building, and some people are saying we ought to evacuate the building. I think we have our priorities sadly wrong.

Also, in response to petitions by the Action on Smoking and Health (ASH), OSHA has already clearly stated their position on environmental tobacco smoke (Table 5). They've stressed they cannot make a rule in the workplace until there is proof of risk in the workplace. Three times in court filings OSHA have stated that the evidence of spousal smoking is not relevant. In all the EPA studies done on spousal smoking in homes, there is insufficient evidence of any risk in the workplace. And before OSHA introduce regulations, they must prove what level of a suspected carcinogen causes risk, and this information is simply missing at this time. All workplace studies thus far show no evidence of increased risk.

What is OSHA supposed to do in this situation? Well, they are already moving towards including environmental tobacco smoke as a typical indoor pollutant. To deal with all indoor pollutants, the best possible approach is a Building Systems Approach. This is not new or unique. It's simply the use of appropriate design criteria, documentation of that criteria, and compliance with design specifications throughout the life of the building. .

All the standards necessary already exist (Table 6). For example, ASHRAE have specified standards for ventilation, comfort, filtration, commissioning, operation and maintenance. In our recommendations to our many hundreds of clients, we point out

to them that if they apply these standards, they will be able to accommodate all indoor pollutants in their particular buildings.

Frequently, we are asked specifically to deal with the issue of smoking. Our first option to any of our clients, regardless of whether they have smokers or not in the building, is that they should adopt a Building Systems Approach and ensure that the workplace conforms to ASHRAE 62-1989. This stipulates that offices need a minimum of 20 cubic feet per minute of outside air per person. Also, filtration should be a minimum of 40 percent efficiency. ASHRAE points out that this will accommodate a moderate amount of smoking and we totally endorse that. In fact, if we look at all the buildings that we have investigated meeting ASHRAE 62-1989 where discretionary smoking is allowed, all the significant pollutants that have been associated with tobacco smoke fall in a range far below even the most stringent guidelines that have been introduced.

We've already seen the examples of benzene, but it's the same for nicotine, respirable particulates, or carbon monoxide. The fact is, if the buildings are run according to the proper ventilation rates, you can definitely accommodate a moderate amount of smoking without any problem whatsoever (Table 7).

Now I am also aware that certain areas of this country have introduced separate smoking areas in buildings and we often counsel our clients that this may be something they could do to meet local regulations or for social or political reasons.

The first option is to separate the smokers and non-smokers. If it's done judiciously, it can be done without any physical barriers and with no problems whatsoever. Where practical, say in cafeterias, we would suggest congregating the non-smokers near the supply diffusers and the smokers directly adjacent to the exhaust or return system in the building. Because you exhaust air from the smoking area, you create a negative pressure in that area. Water cannot flow uphill, nor can air flow against the pressure gradient. The air moves from the non-smoking area to the smoking area. It is taken out of the room, filtered and then diluted with outside air. The air returned to the non-smoking area is perfectly satisfactory.

Indeed, you can take one step further than this. You could introduce in the smoking area a designated filtration system such as electrostatic precipitators, HEPA filters, or activated charcoal to remove odors. In our experience, when these have been used, the air in both the non-smoking and smoking area is way beneath any existing occupational exposure standards and everybody is perfectly happy.

Of course, we can go further. Under certain conditions, we also counsel our clients to consider designated smoking area with their own ventilating system. But the

ventilating system is one of exhaust ventilation. The principle of exhaust ventilation is very simple. If you have a smoking lounge, you fit that with a dedicated exhaust to the exterior of the building. The smoking lounge should exhaust 60 cfm per person, an ASHRAE recommended rate for a designated smoking lounge.

As a result, the smoking lounge now runs at a lower pressure than any adjacent areas, which means that if there are any leaks into that lounge (say somebody opens the door), no smoke can come out of the lounge, air can only pass into the lounge.

In practice, we can have a designated smoking lounge with its own air supply and the exhaust. You size it so that the exhaust is larger than the supply, which means you have a negative pressure and as a result, any adjacent air is transferred from outside the lounge, into the lounge. Indeed, you do not even need doors. The best possible example I can site of this would be Salt Lake City Airport where in the Delta terminal you will see at least three of these lounges, each designated to house 100 people. There's no front on these lounges and everyone in the building congratulates the designers on how well they accommodate smokers. This example is in a Mormon district, a group who obviously abhors smoking, but they are astute businessmen and thus they wish to accommodate passengers using their airport.

I can also point out that when one wants to size lounges for smokers, it is not difficult. In fact, if you look at a typical building with a floor of 10,000 square feet, the average staffing on that floor using national averages would be 70 people. Assuming 30 percent of these people are smokers, that's 21 people. If 50 percent of these smoke at any one time, we would designate a lounge sized to house 10 people. Using ASHRAE criteria, 15 square feet per person in a smoking lounge tells us our lounge should be 150 square feet. This is a typical small 12'x12' office. Note, 150 square feet represents less than 2 percent of the area of this floor, so I think you would appreciate the utilities involved in less than 2 percent of the area are not that significant. Indeed, the only new thing we need to fit in most buildings is an exhaust fan. An exhaust fan sized at 60 cfm per person for 10 persons means 600 cfm. I can assure you that the cost of that fan operation, even if it were to run 365 days per year, 24 hours per day using Washington, DC electricity rates would be less $50 per year.

Finally, I would like to return for one moment to the overall position on indoor air quality. As part of our research in studies of different countries around the world, we have retained the services of Harris Research, the opinion pollster. They conducted totally independent interview polls from 1989 to 1992 of office workers in ten different countries. Nearly 15,000 people have been interviewed in all. Two specific items of these polls are of interest in this debate.

The first is the frequency of smokers in the work area. In the US and Australia they have so significantly reduced the incidence of smoking in the workplace (through social pressure, political reasons or legislation), that only 18 percent of the people in the buildings admit that they were exposed to tobacco smoke. So the air in so many offices is now essentially smoke free. (This figure compares with the other eight countries where, on average, 50 percent of the population say they are exposed to other people's smoke.) The restricted smoking policy was introduced apparently with the goal of cleaning up the indoor environment, hopefully it would reduce absenteeism and improve productivity. The question is, has it worked?

Australia and the USA are two areas with the lowest smoking incidences. However, when we look at how many times people take time off work from problems due to sick building syndrome. Australia and the US virtually top the league of all these different countries. Despite the fact that they've so successfully reduced the instance of smoking, they have done nothing about reducing the real incidence of sickness and absenteeism.

The fact is that that they've reacted to one visible pollutant without realizing what the fault has been. Australia and the US have far more multi-story buildings that are tightly sealed and as a result, all indoor pollutants have been trapped. They have reacted only to the visible pollutants. They've missed the source of the problem, which was inadequate ventilation and faulty building operation and as a result, they've still not addressed the real problem.

If you wish to deal with indoor air issues, you have to address the cause of the problem (Table 8). You have to improve ventilation, filtration and hygiene standards. If you do, you adequately deal with all indoor pollutants, including tobacco smoke. Moreover, with this policy absenteeism drops and productivity is improved.

Thank you very much for your time today, ladies and gentlemen.

Table 1

HBI EXPERIENCE 1980-JANUARY 1993
Major Factors Found
Database: 953 Buildings, 147 Million Square Feet (14 Million Square Meters)

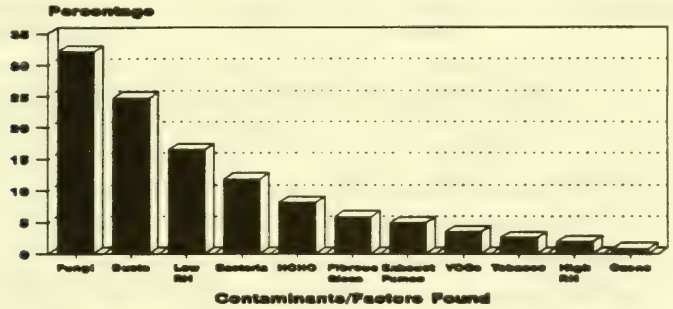

Table 2

HBI EXPERIENCE 1980-JANUARY 1993
Major Contributors to Sick Building Syndrome
Summary

Database: 953 Buildings, 147 Million Square Feet (14 Million Square Meters)

Poor Ventilation	51.5%
Including No Ventilation	19.6%
Inefficient Filters	57.0%
Including Careless Installation	15.1%
Excessively Dirty Ventilation Systems	42.8%
Including Contaminated Ductwork	10.6%
Well Ventilated, Efficiently Filtered, Clean and Well Maintained Systems	39.7%

Table 3

SIGNIFICANCE OF AIRBORNE CARCINOGENS

Example — Benzene

Standards: OSHA PEL 1 ppm (TWA 8 hr)
ACGIH TLV 10 ppm (TWA 8 hr)

Literature Search of all Published Studies (Holcombe 1993)				
Location	Number studies	Number sites	Benzene concentration (ppm)	
			Smoking	Nonsmoking
Offices	9	39	0.0025	0.0020
Homes	7	820	0.0042	0.0025
Transport	3	383	0.0024	0.0092

Benzene concentration: Transport greater than Homes, Homes greater than Offices

Table 4

SIGNIFICANCE OF AIRBORNE CARCINOGENS

Example — Asbestos

Standards: OSHA PEL 0.2 fibers/cc of air (8 hr TWA)

What does this mean?

Consider a 100,000 square foot building
Volume, typically = 1,000,000 ft^3
= 28,320 m^3
= 28,320,000 cc

At 0.2 fibers/cc = 5,664,000 fibers

Thus, 5 million fibers of airborne asbestos
would be acceptable in building

Table 5

OSHA'S POSITION ON ETS

In Response to Petitions by ASH

- Cannot make a ruling in workplace until proof of risk in workplace
- Three times in court filings OSHA have stated that the evidence of spousal smoking is not relevant — they have insufficient evidence of workplace risk
- Before OSHA regulation they must prove what level of suspect carcinogen causes risk in workplace, i.e., quantitative risk information essential

Table 6

BUILDING SYSTEMS APPROACH

In Response to Petitions by ASH

Relevant Guidelines

ASHRAE 62-1989	Ventilation
ASHRAE 55-1981	Comfort
ASHRAE 52-70	Filtration
ASHRAE 1-1989	Commissioning
ASHRAE 4P-1992	Operating and Maintenance

Steps:

1. Design Criteria
2. Construction Techniques
3. Commissioning Process
4. Operator Training
5. Preventive Maintenance
6. Monitoring and Documentation

Table 7

SMOKING POLICIES

Discretionary Smoking — Actual Values

When ASHRAE 62-1989 Ventilation Standards are Met:

HBI's Average Office Measurements		
Substance	Range	Most Stringent Guideline/Standard
Carbon Dioxide (ppm)	400-700	1000 (ASHRAE)
Carbon Monoxide (ppm)	0-4	9 (EPA)
Nicotine ($\mu g/m^3$)	0-8	50 (HBI, ASHRAE)
Benzene (ppm)	0.001-0.002	1 (OSHA)
RSP ($\mu g/m^3$)	20-60	100 (WHO)

Office air quality is excellent and all substances are well within every known standard

Table 8

DEALING WITH INDOOR AIR ISSUES

- Must deal with the whole problem
  Not any single, visible pollutant
- Apply the BUILDING SYSTEMS APPROACH to ensure proper ventilation, filtration and hygiene through DESIGN, CONSTRUCTION, COMMISSIONING, TRAINING, MAINTENANCE, MONITORING and DOCUMENTATION
- This approach adequately deals with all indoor pollutants in the majority of buildings
- The results are REDUCED SICKNESS AND ABSENTEEISM, and INCREASED PRODUCTIVITY

ADDITIONS TO THE RECORD

Society of Critical Care Medicine

TESTIMONY OF JOHN H. HOYT, MD, FCCM ON BEHALF OF THE SOCIETY OF CRITICAL CARE MEDICINE
BEFORE THE HOUSE PUBLIC WORKS AND TRANSPORTATION COMMITTEE
SUBCOMMITTEE ON PUBLIC BUILDINGS AND GROUNDS
TESTIMONY IN SUPPORT OF H.R. 881,
BANNING SMOKING IN FEDERAL AND CONGRESSIONAL BUILDINGS

March 11, 1993

Mr. Chairman and Members of the Subcommittee, I am John Hoyt, MD, FCCM, Chairman of the Critical Care Medicine Department and Medical Director of the Medical/Surgical Intensive Care Unit (ICU) at St. Francis General Hospital in Pittsburgh, Pennsylvania. St. Francis General Hospital is a 750-bed, full-service facility that has provided care to patients in Pittsburgh for 125 years. I am also a clinical professor of anesthesiology and critical care at the University of Pittsburgh, part of the largest fellowship training program of critical care physicians in the world.

I am a board-certified critical care medicine physician. My medical practice focuses on the care of individuals who suffer from immediate life-threatening illness and injury. My patients develop lung, brain and heart failure that is usually a result of trauma, extensive surgery, overwhelming infection, heart attack, stroke and poisoning that occurs from tobacco smoke.

My testimony today is presented as Treasurer of the Society of Critical Care Medicine, an organization comprised of more than 7,500 physicians, nurses and allied health care practitioners who are committed to the care of the approximately 4 to 6 million Americans who become critically ill or injured each year. Members of the Society are, unfortunately, all too well aware of the significant health care consequences of prolonged smoking or exposure to second-hand smoke that cost this country significant health care resources each year. Smoking kills nearly 400,000 Americans annually, including 115,000 from coronary artery disease; in general, smokers double their risk of heart disease compared to non-smokers; while those smokers between the ages of 45 and 64 years of age are three times more likely than non-smokers to suffer from heart disease. Another 27,000 Americans die from stroke, 136,000 from cancer and 50,000 from other related illnesses. On any given day, 14.6 percent of all ICU admissions are diagnosed with respiratory insufficiency or failure. Every year, $22 billion are spent on the medical costs related to illnesses caused by the addiction to tobacco. Medicare and Medicaid spend $4.2 billion annually for the care of patients with tobacco-related illnesses.

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Two weeks ago, I experienced one of the busiest nights in the ICU I can recall in my 17-year career as a critical care intensivist. Of the eight patients I admitted to the ICU that night, six were admitted with smoking-related illnesses and needed the support of a ventilator to breathe. Their disease, Chronic Obstructive Pulmonary Disease (COPD), literally obstructs the workings of the lung. The lung consists of a delicate architecture of tubes and sacs, that allow gas exchange and oxygen delivery. Smoking leads to damage of these tubes and sacs and obstructs the flow of oxygen to the lung. COPD causes inadequate function of the lung, shortness of breath, limited exercise ability, the need for support from mechanical ventilation and finally death from respiratory failure. The process is long and debilitating; many COPD patients suffer for as long as five years before dying of this disease. Their care is expensive; the cost for a patient to receive support from a ventilator is eight times more than average hospital care. Sixty thousand Americans die each year of Chronic Obstructive Pulmonary Disease.

How do tobacco-related costs add up in the ICU? My colleague, Dr. Timothy Buchman, Co-Director of the Surgical ICU at Johns Hopkins Hospital in Baltimore, recently published a study of admissions to his unit which showed that tobacco, alcohol and illegal drug-related illnesses and injuries accounted for 28 percent of the admissions and 29 percent of the medical costs during a 15-week period. The 435 patients admitted to this particular ICU generated $3,014,953 in costs in a unit with a per night cost of $3,000 per bed. The 59 patients with tobacco-related illnesses alone accounted for $481,648 in costs, or approximately $8,000 each. Tobacco-related cases that involved cancer and respiratory diseases accounted for 14 percent of the admissions and 16 percent of the costs. All but one of the 59 cases were related to cigarette smoking; one case was related to cigar smoking.

The physical and societal ills associated with smoking affect not only adults, but our children as well. A 1991 report from the Department of Health and Human Services reported that half of all eighth graders and nearly two-thirds of all tenth graders have tried cigarettes. Infants are also adversely affected; 20 to 30 percent of low birth weight babies, up to 14 percent of preterm deliveries and approximately 10 percent of all infant deaths are attributable to cigarette smoking during pregnancy.

The life-threatening effect of passive smoking has been well documented. The recent EPA report, "Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders" clearly stated the dangers of breathing second-hand smoke. When inhaled, environmental tobacco smoke is a human carcinogen that can be directly blamed for the lung cancer deaths of 3,000 non-smokers each year in the U.S. In children, second-hand smoke clearly increases the risk of lower respiratory tract infections, including bronchitis and pneumonia, resulting in the hospitalization of 7,500 to 15,000 infants and children each year. This carcinogen also increases the prevalence of middle ear disease and the frequency and severity of symptoms in asthmatic children. According to the EPA, the condition of 200,000 to 1 million

asthmatic children is worsened by exposure to second-hand smoke. The Society of Critical Care Medicine agrees with former EPA Administrator William K. Reilly's statement that environmental tobacco smoke may present the most important environmental health risk facing Americans today.

In this era of historic health care reform, where quality of care, access to care and cost of care are such intensely debated issues, these results - - which are due to ***preventable*** illness - - are entirely unacceptable. The "common courtesy" approach to eliminating passive smoking that is advocated by the tobacco industry has not worked. Policymakers, health care providers and patient advocates, all must ensure that the health of Americans is not further endangered by tobacco smoke. We must ensure that scarce and expensive health care resources are allocated in the most efficient manner possible. Too many other *unpredictable and unpreventable* illnesses and injuries require our attention, including overwhelming infection, traumatic injury, multi organ system failure and others. Improving the health of all Americans, by eliminating smoking and the diseases it causes, will prolong life and release the precious human, physical and financial resources needed to care for other patients.

The Society of Critical Care Medicine supports H.R. 881, which would establish a complete ban on smoking in all federal and Congressional buildings. Serious health problems caused by smoking have been well documented over a number of years. We believe it is time for Congress to take a stand and take the lead on this important issue by banning smoking in all private offices and public areas of federal and Congressional buildings.

Mr. Chairman, members of the Subcommittee, thank you for the opportunity to present the Society's views on this very important issue. We look forward to working with Congress and Members of the Subcommittee to eliminate this life-endangering threat to the lives of all Americans, adults and children. I will be happy to answer any questions you or the Subcommittee Members may have at this time.

American Heart Association

AMERICAN LUNG ASSOCIATION®
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Coalition on Smoking OR Health

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March of Dimes Birth Defects Foundation

TESTIMONY OF ALFRED MUNZER, M.D.

ON BEHALF OF

THE COALITION ON SMOKING OR HEALTH

to the

House Committee on Public Works and Transportation

Subcommittee on Public Buildings and Grounds

U.S. House of Representatives

RE: H.R. 881, LEGISLATION TO BAN SMOKING IN FEDERALLY OWNED AND LEASED OFFICE BUILDINGS

March 11, 1993

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Mr. Chairman, and members of the subcommittee on Public Buildings and Grounds, I am Dr. Alfred Munzer, President-elect of the American Lung Association (ALA). I am also Director of Critical Care and Pulmonary Medicine at Washington Adventist Hospital in Takoma Park, MD, where I specialize in the treatment of diseases of the lung.

The ALA is the nation's oldest voluntary health organization and is dedicated to the prevention and control of lung disease. This organization, and its medical section, the American Thoracic Society, has long recognized the contribution of indoor and outdoor air pollution to the development and exacerbation of lung disease. The ALA has devoted the past 26 years to the implementation of programs aimed at improving air quality in our homes and in our communities.

Today I am testifying of behalf of the American Lung Association, the American Cancer Society, and the American Heart Association, united as the Coalition on Smoking OR Health. Formed in 1982, the coalition has worked to heighten public awareness about the impact of tobacco consumption upon public health. It believes strong measures should be imposed to discourage tobacco use in all segments of the population, including youth, women, and minorities.

As a pulmonary physician, I all too often see first had the devastation cased by tobacco use. I see the men and women who come to me with end-stage lung cancer or emphysema, seeking a medical miracle to cure their disease. I see the children who cough and wheeze as their asthma is made worse by exposure to smoke exhaled by smokers and that comes from the burning end of a cigarette, pipe, or cigar. Smoke of this nature is commonly called involuntary, passive, or secondhand smoke. However, more recently, it increasingly referred to as "ETS", or environmental tobacco smoke.

I cannot express to you how critical it is for us to respond to the ETS issue. Conclusions drawn from the Environmental Protection Agency's (EPA) risk assessment on ETS reenforces the sense of urgency in this regard. If we do not take immediate action, or ignore its impact on public health, ETS can easily be the cause of approximately 3,000 lung cancer deaths in nonsmokers in the coming year. I am certain this is not the future trend our society desires to establish.

Today, I do not intend to argue whether smokers should have a right to smoke it public, nor am I here to urge the subcommittee to revoke this privilege. However, I have elected to appear before you due to ongoing concern regarding the health effects of ETS for nonsmokers and particularly children, and the need to impose stringent measures, both in

government and the private sector, to adequately address this growing public health concern.

I want to begin by reflecting on the evidence which has supported and recently led to the EPA findings on ETS, and move into a examination of the methodology which supports this agency's assertion that ETS is a carcinogen. I will speak briefly to the claims raised by tobacco advocates regarding the validity of the EPA findings, and lastly, focus on public interest in and support for significant action to limit or eliminate exposure to ETS is public areas.

ETS has been the topic of discussion for more than 20 years. Its health effects were first reviewed in 1972 in the U.S. Surgeon General's report on smoking and health. That report was devoted, in part, to public exposure to air pollution caused by tobacco smoke. It concluded that "an atmosphere contaminated with tobacco smoke can contribute to the discomfort of many individuals."

In 1982, the U.S. Surgeon General <u>again</u> examined the issue of passive smoking, but this time in the context of smoking and the development of cancer. At that time there were only 3 epidemiologic studies linking passive smoking and lung cancer. Even with this limited amount of evidence, the Surgeon General concluded that the evidence in these studies

is the cause for serious concern regarding the possible serious public health problem associated with passive smoke and lung cancer.

By 1986, federal interest in the health effects of ETS had grown to the extent that the U.S. Surgeon General released a report devoted entirely to the issue of passive smoking. By that time, the number of epidemiologic studies had increased to 13, 11 of which showed a positive correlation between passive smoking and lung cancer in healthy nonsmokers. Based upon these findings, the Surgeon General concluded that exposure to secondhand smoke is a cause of lung cancer in healthy nonsmokers. He also concluded that children whose parents smoked had an increased frequency of respiratory symptoms and infections, compared to children whose parents were nonsmokers.

Several private organizations -- the National Academy of Science and the International Agency for Cancer Research -- published reports which drew conclusions similar to those of the EPA. The International Agency for Cancer Research, for example, released a report on cancer which concluded that "knowledge of the nature of sidestream and mainstream smoke, of materials absorbed during passive smoking, and of the quantitative relationships between dose and effect that are commonly observed from exposure to carcinogens leads to the

conclusion that passive smoking gives rise to some risk of cancer."

Shortly after the release of these studies, the EPA began to examine the health effects of passive smoking on children and adults. The agency issued an initial analysis of the risks of exposure to ETS in May, 1990. Entitled, "Health Effects of Passive Smoking: Assessment of Lung Cancer in Adults and Respiratory Disorders in Children," the risk assessment focused on the potential correlation between ETS and lung cancer in nonsmoking adults and respiratory disease and pulmonary effects in children.

On January 7, 1993, the EPA released its final report assessing current scientific evidence on the risks of exposure to ETS. Based on the total weight of evidence in the scientific literature, the EPA designated ETS as a Group A carcinogen, a rating used only for extremely hazardous substances known to cause cancer in humans. It ranked ETS in a class of carcinogens which includes asbestos, benzene, and radon.

After evaluating 30 epidemiological studies on lung cancer in nonsmoking adults, the EPA determined that ETS is responsible for approximately 3,000 lung cancer deaths each year. The agency also added that ETS accounts for the development of 20

percent of all lung cancers caused by factors other than smoking. For the average adult, ETS increases their risk of cancer to approximately 2 per 1,000. From these conclusions, it is clear that ETS is far more hazardous to the health of nonsmoking adults.

After evaluating more than 100 studies on respiratory health in children, the EPA concluded that ETS exposure increases their risk of lower respiratory infections, like bronchitis and pneumonia. ETS is known to cause an estimated 150,000 to 300,000 cases of respiratory illnesses in children up to 18 months each year. Of these cases, 7,500 to 15,000 result in hospitalization.

ETS exposure is also associated with additional attacks and increased severity of symptoms in children with asthma. The EPA estimates that 200,000 to 1 million asthmatic children have their condition worsened by ETS, and that ETS is a risk factor for new cases of asthma in children without a history of symptoms.

Also of concern are the risks for children whose mothers smoked during and after pregnancy. The U.S. Department of Health and Human Services has reported that, under these circumstances, children are three times more likely to die of Sudden Infant Death Syndrome (SIDS) than children of

nonsmoking mothers. The risks of SIDS double for children whose mothers smoked after birth and not during pregnancy than for children reared in nonsmoking environments.

The evidence presented represents very sound science and more than adequately supports the conclusions by the EPA regarding exposure to ETS. Uniquely, each of the studies and reports used to reach this conclusion were developed and edited by different processes. In contrast to assertions made of opponents of the EPA's findings, such as those offered by the tobacco industry, it is this diverse methodology which only strengthens the validity of the conclusion of this research combined.

Without spending too much time on the tobacco industry's criticisms of the risk assessment, let me first remind the subcommittee that after 60,000 studies linking smoking with disease and death, this industry still fails to acknowledge that it produces a lethal product. This is an industry which has criticized each Surgeon General's report since 1964. Among the industry criticisms is the failure of the EPA to include studies which show no relationship between ETS and lung cancer. Among the studies cited by the industry as examples are several funded by the National Cancer Institute:

♦ Brownson, PhD., et.al. Passive Smoking and Lung

Cancer in Nonsmoking Women. Am J Public Health 82:1525-1530, 1992.

This study was published in November 1992, too late for inclusion in the risk assessment unless it was further delayed. The industry contends that the risk assessment would change if the study were included. However, the author's of the study conclude: "Ours and other recent studies suggest a small but consistent increase risk of lung cancer from passive smoking. Comprehensive actions to limit smoking in public places and worksites are well-advised."

♦ Stockwell, Sc.D., et.al. Environmental Tobacco Smoke and Lung Cancer in Nonsmoking Women. J Natl Cancer Inst 84:1417-1422, 1992.

This study was not included in the final risk assessment and again the industry claims it is a negative study therefore left out purposefully. However, the author's conclude:

"These findings suggest that long-term exposure to environmental tobacco smoke increases the risk of lung cancer in women who have never smoked."

The real issue here is statistical significance and how it is used. In defining the true meaning of statistical

significance, I'd like to defer to the description used by a well-noted environmental epidemiologist, Dr. Douglas Dockery, and Associate Professor at the Harvard School of Public Health. Dr. Dockery suggests:

> " A naive critique would say that those studies which are not 'statistically significant' do not show an effect. However, statistical significance is not a measure of association or environmental tobacco smoke with lung cancer, but rather a measure of the stability of the association. It measures the statistical power of the study. In a crude sense it is a measure of study size, and studies that do not achieve statistical significance are simply too small. This does not mean that they do not provide important information on risks. It is not appropriate to discard studies which do not achieve statistical significance, but rather they should be included giving them a weight which reflects the stability, that is the uncertainty, of their effect estimate. This is exactly what the meta-analysis of these studies provides."

Mr. Chairman, we at the Coalition on Smoking OR Health believe the EPA's findings are clear, objective, and complete in regard to ETS. The evidence used to show the relative risks associated with exposure to ETS, and its linkage to the development of lung cancer, are more compelling than similar

correlations drawn for other environmental carcinogens. I hope the evidence I have presented to the subcommittee today will enable you to step beyond the criticisms offered regarding the validity of the EPA risk assessment, and encourage to you move forward in your efforts to address the real issue on the table -- adequately responding to the public health issue associated with exposure to ETS, particularly in those places where people spend a lot of time.

Let me commend the subcommittee for the effort and commitment it has made to this issue, thus far. The introduction and careful review of legislation to ban smoking in federal buildings is a very important step in this process. The Coalition on Smoking OR Health supports the bill -- H.R. 881 -- on which this hearing is being held today, and similar proposals offered by other Members of Congress, from whom you will hear later.

In examining the proposal to ban smoking in federal buildings, let me remind you that the federal government has taken little initiative to protect federal workers from exposure to secondhand smoke. Any action taken to date remains inconsistent with each federal agency responsible for its own policy. The General Services Administration (GSA) is reconsidering its regulations, but GSA space accounts for

only 10 percent of federal building space.

I urge this subcommittee to take into consideration growing interest in smoke-free public places, which has gained momentum since the release of the EPA risk assessment. Based on a public opinion survey conducted by the American Lung Association, it is clear that more and more Americans believe ETS is harmful, and that they prefer smoke-free public places, as opposed to those environments in which smoke is permitted.

In this survey, the Lung Association found that 8 in 10 smokers know that ETS is bad for the people around them. We also found that nonsmokers are more likely than smokers to strongly agree about the harmful effects of ETS exposure. The survey found increasing support for total bans on smoking in public places such as restaurants, workplaces, hotels, buses, and trains. And though current smokers are more likely than others to believe that smoking in public places should be restricted, very few smokers surveyed favored no restrictions.

Clearly, as the awareness of the health hazards of ETS increases, more Americans are striving to live, work, and breathe in smoke-free environments. Very few of us make it through each day without exposure to ETS. Those who are

confined to indoor environments -- like employees and staffers in the House of Representatives -- are no exception. It is unfortunate that the House Building Commission's recent decision to merely restrict smoking to certain areas does not provide the adequate protection needed.

I believe the 1986 report of the Surgeon General has the best recommendation for us to consider. In its conclusion, the report clearly states, "Simple separation of smokers and nonsmokers within the same air space may reduce, but does not eliminate, exposure of nonsmokers to ETS." Therefore, it is the responsibility of employers and employees to "ensure that the act of smoking does not expose the nonsmoker to tobacco smoke" and for smokers to "assure that their behavior does not jeopardize the health of other workers." In addition, the Surgeon General stated that nonsmokers have the "responsibility to provide a supportive environment for smokers who are attempting to stop."

The House of Representatives owes its employees and the people of this country who frequent the Capitol grounds to provide a healthy and safe environment. By going smoke-free, the House will contribute to the notion that nonsmoking is a social norm.

On behalf of the American Lung Association, and other members

of the Coalition on Smoking OR Health, I would like to thank you for the opportunity to testify before the subcommittee on the impact of ETS exposure upon public health. Again, we urge you to step beyond the criticism offered regarding the validity of the EPA data and take into account the points we have raised today, which demonstrate a need for government action. Please know you have our organization's support and encouragement as you continue to review this very pressing issue.

TO PROHIBIT SMOKING IN FEDERAL BUILDINGS

THURSDAY, APRIL 22, 1993

HOUSE OF REPRESENTATIVES,
SUBCOMMITTEE ON PUBLIC BUILDINGS AND GROUNDS,
COMMITTEE ON PUBLIC WORKS AND TRANSPORTATION,
Washington, DC.

The subcommittee met, pursuant to notice, at 9 a.m., in room 2253, Rayburn House Office Building, Hon. James Traficant, (chairman of the subcommittee) presiding.

Mr. TRAFICANT. The hearing will come to order on H.R. 881.

I'd ask the distinguished gentlewoman from the District of Columbia if she has an opening statement she'd like to make?

Ms. NORTON. Mr. Chairman, I'd ask that my full opening statement be put in the record.

I just want to commend the chairman for his tenacity in remaining with an issue that is of great importance and one that needs a strong hand. Those of us who survive the smoke will live to thank you for what you're doing with this bill.

[Ms. Norton's prepared statement follows:]

STATEMENT OF CONGRESSWOMAN ELEANOR HOLMES NORTON

Ms. NORTON. Chairman Traficant, I am pleased to join you and my distinguished colleagues at this hearing to consider H.R. 881, a bill to prohibit smoking in federally owned and leased buildings. I applaud you, Mr. Chairman, for your continuing interest in protecting federal workers from the negative effects of secondhand smoke, and welcome the witnesses who will appear before us this morning.

In spite of the fact that tobacco smoking has long been recognized as a leading cause of disease and death, 25.7 percent of Americans—46.3 million people—smoked in 1991. Many of these people do not stop their smoking at the workplace, and, consequently, their co-workers have been, and continue to be, exposed to secondhand smoke. In recent years, medical and environmental studies have revealed that nonsmokers, who are exposed to smoke exhaled by smokers, may be at risk for developing certain negative health effects associated with somking.

In recognition of the dangers of passive smoking, there has been widespread implementation of policies to protect non-smokers. For instance, no longer can a person smoke on an airplane during a domestic flight and in some cities, such as New York, there is a total ban on smoking in municipal government buildings. By prohibiting all smoking in federally owned and leased buildings, H.R. 881 continues this sound practice of establishing smoking policies that reflect health and scientific findings on the risks of secondhand smoke.

H.R. 881 deserves our support. It is a cost-efficient measure that will provide federal employees with a healthy, smoke-free work environment as they provide the American people with faithful service. Again, my thanks to Chairman Traficant and to our witnesses here today.

Mr. TRAFICANT. I thank the gentlewoman from the District of Columbia.

Before we proceed with our first panel of witnesses, I would like to insert into the record at this point a statement from the Architect of the Capitol, Hon. George M. White.

[Mr. White's prepared statement follows:]

STATEMENT OF GEORGE M. WHITE, FAIA, ARCHITECT OF THE CAPITOL

Mr. Chairman, I am pleased to offer this statement for the record for the Subcommittee's consideration on H.R. 881, a Bill to ban smoking in all Federal Buildings.

The question of banning smoking in the Capitol Complex is, of course, a matter for consideration by the Speaker, House Office Building Commission, and the Senate Committee on Rules and Administration, the entities that have jurisdiction over the Capitol buildings.

In accordance with our previous discussions on this matter, I am pleased to advise the Subcommittee about the ventilation systems in the buildings, and the costs associated with the elimination of recirculated air in designated smoking areas.

Each of the Capitol buildings is different in size and arrangement of space since they were constructed at different times in history and often for distinctly different purposes. In practically every instance, hearing rooms are provided with a separate air handling unit that serves only those spaces. However, office spaces are served by central air handling units. Each of these air handling units receives and distributes outdoor air and recirculated air in an adjustable amount. The outdoor air quantity is usually fixed during the cooling months and therefore, the recirculated air is also fixes; however, the quantity of outdoor air is usually a variable during the heating months because of the extremes of outdoor temperature variations. The outdoor air quantity could vary from 20% to 100% of supply air during the heating months since it is usually controlled by a mixed air thermostat set at approximately 50F.

The outdoor air and recirculated air are mixed and the resultant mixed air is filtered. However, the filter medium is not specifically intended to remove smoke particles. If that were required, either a very dense type (HEPA) filter or an electrostatic precipitator type filter would have to be installed. These would be expensive to install and to maintain and are generally not 100% effective.

The House Chamber has a typical air handling unit that supplies a mixture of outdoor air and recirculated air. The Gallery is supplied from a separate unit that also supplies the adjacent Cloakrooms. However, all air supplied to the Cloakrooms is exhausted and not recirculated through the Gallery air handling unit.

Increasing the quantity of outdoor air introduced by each air handling unit, just to increase dilution of recirculated air at all times, would cause a substantial increase in energy consumption. Rather, it is more logical to control air contaminants (such as smoke) at the source. Separate rooms could be set aside as smoking areas so that all of the supply air could be exhausted and not recirculated into other occupied spaces.

In order to provide an appropriate environment within any space designated as a smoking area, an exhaust air system must be available that directly transmits the contaminated air to outside the building and not to the recirculation air system. Toilet rooms, typically, have exhaust air systems that could accommodate this function, if necessary, with relatively minor modifications. However, if other existing spaces are designated smoking areas, a new exhaust system must be designed and installed based upon the size and location of the space within each building. The cost of a new exhaust system for such a space could vary from $2,000 to $10,000 with an average cost per space of approximately $5,000. We would be able to develop more definitive cost estimates after the specific "no smoking spaces" are identified and design criteria completed.

Mr. Chairman, that completes my prepared statement and I will be pleased to discuss this matter in more detail if that is deemed desirable.

Mr. TRAFICANT. Our first two witnesses are distinguished professionals from the health field, Dr. John W. Hoyt, Chairman, Department of Critical Care Medicine, St. Francis Medical Center, Pittsburgh, Pennsylvania and Dr. Alfred Munzer, President, American Lung Association, Washington, D.C.

We will start with Dr. John W. Hoyt. We welcome you, Doctor. It's good to see you.

TESTIMONY OF DR. JOHN W. HOYT, CHAIRMAN, DEPARTMENT OF CRITICAL CARE MEDICINE, ST. FRANCIS MEDICAL CENTER AND SOCIETY OF CRITICAL CARE MEDICINE; AND DR. ALFRED MUNZER, PRESIDENT, AMERICAN LUNG ASSOCIATION

Dr. HOYT. Thank you very much.

Mr. Chairman and members of the subcommittee, I'm here representing the Society of Critical Care Medicine, which is an organization of 7,500 physicians, nurses and respiratory therapists that work in the intensive care units across the country. I'm also here representing St. Francis Hospital in Pittsburgh, Pennsylvania.

What I would like to do is to try and represent my personal experience from the intensive care unit. I am a critical care physician, which means that I have devoted my career to caring for patients in the intensive care unit. That is an area of the hospital where there is a very substantial number of patients who are admitted with the complications of smoking.

On a recent evening call where I had eight admissions to the intensive care unit, six of those patients were in respiratory failure and all six of them ended up on a breathing machine. In each case, those patients were ones who had respiratory failure that either totally attributable or partially attributable to the long-term effects of smoking.

In previous hearings that I have been a part of, I have noted that there is a major emphasis in the discussion that's taking place on the relationship between smoking and cancer. I don't think I can particularly add anything to that. Instead, I would like to address the issue of the relationship between smoking and heart disease and smoking and chronic obstructive pulmonary disease.

Smoking and cancer is certainly there. It is certainly a major problem because most people don't live much more than one or two years after the diagnosis of lung cancer. Smoking in coronary artery disease is an even bigger issue. Coronary artery disease is at epidemic proportions in the United States today and smoking and chronic obstructive pulmonary disease is also a major issue because patients tend to live 4 or 5 years after the diagnosis of chronic obstructive pulmonary disease. They have many, many admissions to the hospital; and it turns out to be a very debilitating illness and a very, very expensive illness because of multiple admissions to the intensive care unit.

I have submitted to the committee several articles. The first one I would like to refer to relates to the issue of smoking and coronary artery disease. It appeared in January of 1993 in the Journal of the American College of Cardiology. It comes from San Francisco, California. It is a rat study.

We've talked a lot in the past here about good science and bad science. This is really quite an excellent article where, in my mind, the science is unquestionable.

They exposed a population of rats to a high dose of environmental smoke, not smoking rats but just smoke in the environment and they exposed rats to a low dose of smoke which would be equivalent to the amount of smoke that you might experience when air circulates in a building where people are allowed to smoke freely in various sorts of areas.

In both of those populations of rats, there was a tremendous acceleration in the hardening of the arteries, the thickening of the vessels, the atheromas, the fattiness of the vessel, which is what causes coronary artery disease in patients today.

In my opinion, this is very convincing evidence, when supported by other evidence from the American Heart Association, that not only smoking directly but sidestream smoking, when you are exposed to it as a nonsmoker, passive smoking, contributes to the progression of hardening of the arteries, coronary artery disease, chest pain, and ultimately myocardial infarction.

The second issue that I would like to address is the issue of chronic obstructive pulmonary disease. As I had said before, the lung is a very, very delicate life organ that relies on open, clear tubes and sacs in order for oxygen in the air to be picked up by the blood and carried out to meet the metabolic needs of the body.

If these tubes become at all diseased or obstructed, as they do in chronic obstructive pulmonary disease which is a direct complication of smoking, then you have a state of chronic respiratory failure, shortness of breath, inability to move and walk around and a very, very debilitating disease process.

It has been known for some time that smoking causes, in the person that smokes, chronic obstructive pulmonary disease. It has been less clear that sidestream smoking would cause disease, but I would submit, and have submitted to the committee, an article from Chest which appeared in February 1993 looking at the effect of sidestream smoke on pulmonary function in a population of patients who have asthma. They did pulmonary function studies before exposure and after exposure and they noticed that asthma was acutely worsened by the exposure, just sidestream exposure, to smoke.

So smoking from a sidestream or passive type of way is a major problem. It causes coronary artery disease, it aggravates lung disease, and as identified before, it has now been associated with an alteration in the incidence of lung cancer. Certainly lung cancer is a major issue and I don't mean to downplay it but I did want to highlight the fact that there are other very substantial illnesses that are caused by smoking that have not been addressed to the same degree in the subcommittee hearings.

Overall then, I think that the evidence is really quite clear that smoking, including sidestream smoking, leads to something around $20 or $30 billion dollars in unnecessary health care; that the death rate associated with smoking, premature death rate, is probably somewhere in the range of 400,000 patients per year.

For that reason, I certainly, and the Society of Critical Care Medicine, support House Bill 881 to ban smoking in Federal buildings.

I appreciate being here to make this testimony and would be glad to answer any questions that you might have.

Mr. TRAFICANT. The gentleman from Tennessee?

Mr. DUNCAN. I don't think I have any questions. I'd just thank Dr. Hoyt for being here with us.

I would ask, the very last thing you commented on, you said $20 to $30 billion. Why such a wide range there? That sounds like some figure that somebody has just pulled out of the air. When you have that wide of a range, it sounds almost unreliable.

Dr. HOYT. I agree with that, Congressman. The problem I think is addressed in the third article which I submitted to the committee and that is one from the Journal of Chest also in January of 1993, done by a colleague and fellow critical care physician at Johns Hopkins. This is one written by Tim Buckman entitled, "Substance Abuse-related Admissions to the Adult Intensive Care Unit."

What they attempted to do there was to quantify the percentage of admissions to the intensive care unit that are smoking-related. They settled on a figure that about 14 percent of some 435 admissions to the ICU were caused by smoking-related problems and 16 percent of the costs were caused by that.

If I'm to be totally honest with you and to reflect on this, when you see these sorts of patients, it's never a black and white issue. You know that there is a component of smoking, but there is infection on top of the smoking. In any of those given situations, there are so many multiple factors that it's difficult to tabulate in an accurate way how much of a role was played by smoking and how much was played by other problems.

Mr. DUNCAN. Thank you very much.

Mr. TRAFICANT. The gentlewoman from the District?

Ms. NORTON. Dr. Hoyt, your testimony is compelling. Given what you have seen up close that may tell us much about the effects of smoking, I wonder what the opinion of a medical doctor would be about the proposals we're beginning to hear to tax people so that they are less likely to inflict this kind of intentional harm on themselves, or do you believe it would have that effect?

Dr. HOYT. I'm sorry, could you repeat the question?

Ms. NORTON. Having seen up close what cigarette smoking can do to the arteries and other parts of the body, and seeing people only after the damage has occurred, I would be interested in the opinion of a doctor such as yourself on the proposals we are beginning to see in the Congress that cigarettes be taxed so that people are less likely, or so the supposition is, to inflict this kind of intentional harm on themselves? Do you believe it would have that effect; would the medical community, so far as you know, support that kind of act?

Dr. HOYT. I suspect overall that the medical community would probably support that. The only problem that I have is that smoking is clearly an addiction. It is as much an aspect of substance abuse as any other drug addiction that we talk about. That's why the article from Hopkins is entitled, "Substance Abuse-related Admissions to the Adult Intensive Care Unit."

What that means is that as you raise the price, it puts the feet to the fire to try and stop, but you need to be compassionate to the smokers and also come along with programs that help them to break that addiction because there's going to be a lot of people who are addicted who are going to have a hard time even with substantial prices to break that habit. Particularly for people with limited finances, they are going to continue to be very drawn to the smoking habit and it's going to more and more impinge on their finances for food, shelter and so forth. They are going to need assistance to be able to break this very, very clear addiction.

Ms. NORTON. You don't have medical cures for the effects of smoking. Are we beginning to have medical cures for the addiction to smoking?

Dr. HOYT. I believe within the mental health community, there are programs of the type like Alcoholics Anonymous and there are the nicotine patches which have a decreasing dose that can help. Those patches alone may not be the ideal way to go but with psychological reinforcement and backup combined with those patches, you can very successfully get people off of tobacco when they are willing to do that.

As you know, the Joint Commission for Accreditation of Hospitals has banned smoking in every hospital in the United States. Unfortunately, as you walk in the front door of most hospitals, there is an array of cigarette butts all over the place because everybody goes outside to smoke. However, a lot of hospitals have successfully prepared programs for their employees using psychological counseling and the nicotine patches and have very effectively helped their employees quit the smoking process.

Ms. NORTON. Thank you, Doctor.

Mr. TRAFICANT. While Dr. Munzer takes his seat at the witness table, Mr. Emerson, do you have any questions of Dr. Hoyt?

Mr. EMERSON. Not at this moment, Mr. Chairman.

Mr. TRAFICANT. We will then go forward with the testimony of Dr. Alfred Munzer, President, American Lung Association, Washington, D.C. We'd ask that you summarize, Doctor, rather than read so that we can talk with you.

Dr. MUNZER. Mr. Chairman, members of the subcommittee, I'm Dr. Alfred Munzer, President-elect of the American Lung Association and I'm also Director of Critical Care and Pulmonary Medicine at the Washington Adventist Hospital here in Takoma Park. I specialize in the treatment of patients with diseases of the lung. The American Lung Association is the Nation's oldest voluntary health agency and we are dedicated to the prevention and control of lung disease.

Today, I testify not only on behalf of the American Lung Association but I also speak for the American Cancer Society and the American Heart Association coming together as the Coalition on Smoking and Health.

As a pulmonary physician, I see the ravages of tobacco smoking day in and day out. About 90 percent of the patients I treat have diseases directly attributable to smoking. An increasing number of my patients who do not smoke have diseases that are due to exposure to environmental tobacco smoke.

I cannot express to you how critical it is for us to respond to this issue. Conclusions drawn from the Environmental Protection Agency Risk Assessment on Environmental Tobacco Smoke reinforces the sense of urgency in this regard. If we do not take immediate action, we will fail to prevent 3,000 lung cancer deaths this year alone.

I've elected to appear before you because of ongoing concerns regarding the health effects of environmental tobacco smoke not only in adults but also, and perhaps especially, on children. Their health certainly deserves to be protected.

We've talked about environmental tobacco smoke for over 20 years and its health effects were reviewed as early as 1972 by the Surgeon General who then concluded that an atmosphere contaminated with tobacco smoke can contribute—at that time, what he said was—"to the discomfort of many individuals."

In 1982, the Surgeon General again looked at the evidence and this time, the Surgeon General concluded that there was grave concern regarding the possible serious public health problem associated with passive smoke and the development of lung cancer.

By 1986, the Surgeon General dedicated a whole review to the issue of environmental tobacco smoke and concluded at that time that in addition to the threat of lung cancer, children whose parents smoked had an increased frequency of developing respiratory symptoms and infections compared with the children of nonsmokers.

The National Academy of Sciences and the International Agency for Cancer Research are among the private organizations that added to the body of evidence that concluded that environmental tobacco smoke indeed is very harmful to children especially, but also to adults.

On January 7th of this year, the EPA released its report which placed environmental tobacco smoke among the most hazardous substances in our environment in terms of causing lung cancer. The EPA, as you know, determined that environmental tobacco smoke is responsible for 3,000 lung cancer deaths every year.

After evaluating in addition to that 100 studies dealing with the respiratory health of children, the Environmental Protection Agency also concluded that environmental tobacco smoke is responsible for 150,000 to 300,000 cases of respiratory illnesses among children. Of these, 7,500 to 50,000 result in hospitalization. In addition, environmental tobacco smoke precipitates acute asthma attacks and also may be the underlying cause in the development of asthma.

The evidence presented, we believe, represents very sound science and more than adequately supports the conclusion by the Environmental Protection Agency regarding exposure to environmental tobacco smoke.

I don't want to take too much time to answer the tobacco industry's criticisms of risk assessment, but let me remind the subcommittee that this industry still denies 60,000 studies linking tobacco smoke with death. This is an industry that has criticized the Surgeon General's reports since 1964 and has refused to even acknowledge a possible harm due to direct smoking.

Mr. Chairman, members of the committee, the Coalition on Smoking and Health believes the EPA's findings are clear, objective and complete with regard to environmental tobacco smoke. The evidence used to show the relative risk associated with environmental tobacco smoke and its linkage to the development of lung cancer are more compelling than correlations drawn for other environmental carcinogens.

We encourage you to move forward in your efforts to address the real issue on the table, adequately responding to the public health issue associated with exposure to environmental tobacco smoke. A ban on smoking in all Federal buildings, we believe, is an ex-

tremely important measure in that regard, not just to protect Federal workers and visitors to Federal buildings, but also as an example to the rest of the Nation. Environmental tobacco smoke is a major health hazard; it's a public health issue; and we believe that the bill that you are considering is a very important first step in achieving a healthier society.

Thank you.

Mr. TRAFICANT. Thank you, Doctor.

The gentleman from Tennessee?

Mr. DUNCAN. Very briefly. Do you think there has been a deterioration or an improvement in the indoor air quality over the last two years?

Dr. MUNZER. We at the American Lung Association are very concerned about many factors that are involved in the deterioration of indoor air. I can't say specifically over how many years or whether there has been a deterioration over the last two years or five years.

Environmental tobacco smoke remains the most important form of air pollution, especially among those we can do something about.

Mr. DUNCAN. It's now been almost 30 years since the original Surgeon General's report. Why do you think that we're just now hearing all that we're hearing about the ETS issue?

Dr. MUNZER. I believe that we're confronted by a very powerful lobby, the tobacco industry. The American Lung Association and the other organizations I represent today have been speaking about this issue for a very long period of time, but we do not have the same resources that are available to a very powerful industry. We are delighted that the tide is turning. Smoking has been banned on airlines. There was a fear that this would create tremendous mayhem on the airplanes; that has not happened. I think we are gradually coming to the idea of a smoke-free society.

Mr. DUNCAN. I'm told of the 3,000 deaths that you mentioned, that also includes deaths that occurred from environmental tobacco smoke in the home?

Dr. MUNZER. That's correct.

Mr. DUNCAN. What percentage of those would you attribute to secondhand smoke in the workplace or has it been broken down in that way?

Dr. MUNZER. To my knowledge, that hasn't been broken down, but epidemiologic studies are not the only type of evidence that links environmental tobacco smoke to lung cancer and other diseases. Probably the strongest argument for the hazardous effect of environmental tobacco smoke is simply that the components of environmental tobacco smoke are no different and perhaps more hazardous according to some studies than those inhaled by the smoker who at least filters out some of the substances and retains them in his body. There is no safe threshold for any of these substances that has ever been determined.

Mr. DUNCAN. Since you have been involved with the American Lung Association over the years, have you seen other studies similar to the EPA study that have specifically mentioned ETS and deaths caused by ETA? What I'm wondering is do you think that 3,000 estimate is high, low, extremely conservative, right on target? I'm just curious. Based on other studies that you've seen, have

you seen any other studies that said that deaths were caused by ETS?

Dr. MUNZER. Both the Surgeon General's report in 1986 and the study of the National Academy of Sciences came to a similar conclusion. The estimates that were given in those studies were higher. If I remember correctly, they were in the range of 5,000 deaths caused.

Also, I would like to enter into the record, data released by the American Heart Association last year which focuses on mortality and cardiovascular disease rates in persons exposed to ETS. Much of this data coincides with my testimony given here today. (Position paper: Environmental Tobacco Smoke and Cardiovascular Disease, American Heart Association, may be found beginning on page 411.)

Mr. DUNCAN. Based on everything that you know about this and your estimates, what would you estimate? Do you have a figure?

Dr. MUNZER. I'm not an epidemiologist but I do respect the scientists who are involved in the Environmental Protection Agency study and I accept their figures.

Mr. DUNCAN. All right.

Thank you very much.

Mr. TRAFICANT. The gentlewoman from the District of Columbia, Ms. Norton.

Ms. NORTON. Thank you, Dr. Munzer, very important testimony.

I'm interested in smoking addiction. Dr. Hoyt testified that what we do to discourage smoking needs also to be met by some work to help people to fight the addiction of smoking. Does your work in the American Lung Society indicate that there is substantial research being done to relieve the addiction itself, and if so, what kind of research is being done?

Dr. MUNZER. There is research being done in the area of tobacco addiction, probably not enough considering that this is a problem that affects 25 percent of our population at this time. All the evidence seems to indicate that the addiction to nicotine is at least as strong as the addiction to heroin or cocaine. As a public health problem, this is indeed a huge one.

Mr. Chairman, I ask that a summary of the 1988 Surgeon General's Report entitled, "The Health Consequences of Smoking & Nicotine Addiction" be inserted into the record to supplement my testimony today. (See page 424.)

There have been some advances, as you know, in the treatment of this addiction. We now have some new tools available, including nicotine substitution therapy by means of patches or gum and when these are coupled with a strong behavior modification program, these are effective in helping people quit smoking which is a very important thing indeed. We would hope that as employers restrict smoking in their offices, in their places of work, they put into place smoking cessation programs.

Mr. Chairman, the American Lung Association has designed a program of this nature for the workplace. I ask that our publication on it be inserted into the hearing record. (See page 450.)

We also hope that this will be a part of the health care reform proposal coming down.

Ms. NORTON. I'm glad, of course, to see such efforts, but I must say that compared to the high tech research, if I may call it that,

going on to help in the relief of diseases caused by smoking, this is real low tech stuff when it comes to fighting the addiction itself. We really are putting all of our resources, it would seem, into the back end and now, of course, we're getting harsher and harsher on smokers as we must. We're sending them outdoors and we're perhaps going to charge them more for cigarettes. We have been doing that in any case, but the research on the addiction itself, when you consider how advanced American medical science is, seems almost trivial by comparison.

Dr. MUNZER. Especially when one takes into consideration the total health bill generated by smoking. Smoking costs the United States at least $65 billion each year in health care costs and lost productivity.

Ms. NORTON. I would like to see the American Lung Society and the American Heart Association do more to press for research into the addiction itself because both organizations do such a marvelous job on the back end of this problem.

Finally, what do you believe has been chiefly responsible for the diminished smoking among some and the increased smoking among others in society?

Dr. MUNZER. The major factor that seems to influence smoking, especially among young people, is advertising. There continues to be a tremendous amount of advertising by the tobacco industry which is increasingly targeted to special populations. There are about 1,000 smokers who die every day, 2,000 people who quit every day and they have to be replaced by new smokers. Those new smokers are generated by an advertising campaign, as the Joe Camel campaign has indicated, and is probably the best example of advertising that is targeted to children. This has been well-documented in some very, very worrisome studies in the Journal of the American Medical Association.

So controls on advertising, controls on access to tobacco products for children, we believe is extremely important.

The American Lung Association has a booklet on addressing this issue which I wish to be inserted into the hearing record. (See page 485.)

Ms. NORTON. Is advertising also responsible for women who at some age levels have increased their smoking as well?

Dr. MUNZER. Absolutely. The advertising is targeted to women, it's targeted to African Americans. The industry has done a very good job at segmenting the population and targeting its message.

Ms. NORTON. Thank you, Dr. Munzer.

Thank you, Mr. Chairman.

Mr. TRAFICANT. The gentleman from Missouri.

Mr. EMERSON. I would ask, Dr. Munzer, in response to his last answer, is that not the right of free Americans to advertise, to promote a product? I can understand health concerns that you and others have, but aren't we to have any constitutional rights left? There are a lot of other things that are not good for our health. I happen to be overweight. How are you going to start punishing me if I don't lose weight? You want to punish people who won't quit smoking. What are you going to do? You do have people who smoke, who have a right to smoke. We haven't outlawed it, we don't have prohibition on smoking. I have real serious concerns.

You're getting into a realm here of denying freedom of expression when you want to say ban advertising.

Dr. MUNZER. First, in response to Rep. Emerson's question regarding the right of Americans to advertise, I wish to submit for the record a legal analysis prepared by the American Heart Association on the constitutionality of tobacco advertising bans. (See page 415.)

Secondly, The American Lung Association has never advocated a ban on smoking. We recognize that smoking is addictive and that it is indeed very, very difficult for people to quit smoking. By the same token, we believe that children should be protected from a predatory industry, an industry that really preys on our young children. That's, I think, the whole message of the Joe Camel campaign.

Mr. EMERSON. What are you going to do? Okay, I accept that, I understand your rationale. Now, what are we going to do? Are we going to ban advertising Twinkies because they add to our weight? I would wager—I don't know scientifically, but I would wager—that there are as many health problems in this country related to people overeating as there are to people smoking.

I want to know now how you in the medical community, who are so very, very self-righteous about the smoking issue, what you're going to do about some of these other really, really serious problems such as obesity? You know, this country tried prohibition one time and banning alcohol. We've got probably a lot more problems related to alcohol than we do to smoking and I happen to have some personal familiarity with that subject also. What are you going to do, try prohibition again?

I detect here, I really detect, about as high a level of political correctness coming out here as I do health concerns, political correctness being so en vogue these days.

Mr. TRAFICANT. Will the gentleman yield?

Mr. EMERSON. Of course I'll yield to the Chairman.

Mr. TRAFICANT. We're not here today convened to discuss the ban on advertising.

Mr. EMERSON. That's been injected.

Mr. TRAFICANT. We have an esteemed physician, knowledgeable in respiratory complications and problems, discussing a ban on smoking in Federal buildings, not a ban on smoking nor a ban on advertising.

Mr. EMERSON. I want to reclaim my time.

Mr. TRAFICANT. I wanted to state that for the record. I'd like to proceed forward with the witness.

Mr. EMERSON. I'd like to reclaim my time.

I didn't raise the subject of the advertising ban, the witness raised the subject of the advertising ban. I'm not the one who raised it, he injected into the discussion. Is it inappropriate to comment on that subject?

Mr. TRAFICANT. Certainly not. But I'd like to stay in focus with the bill.

Mr. EMERSON. Is the gentleman going to hold a hearing on banning——

Mr. TRAFICANT. Will the gentleman yield?

Mr. EMERSON. Yes.

Mr. TRAFICANT. We have a very controversial issue. We're being faced with worker compensation claims. I guarantee you that when it gets to a court of law, we're not going to be talking about advertising; we're going to be talking about hard-earned taxpayer dollars going to compensate people subject to all these specific reports about ETS.

I want to move our hearing along around banning smoking in Federal buildings and I agree with you, this brings a myriad of problems, a myriad of political consequences and a whole lot of public scrutiny.

Mr. EMERSON. Mr. Chairman, if I may reclaim my time.

Mr. TRAFICANT. I believe we should go forward.

Mr. EMERSON. If I may reclaim my time, would the Chairman then please, perhaps working with counsel, submit to the members of this subcommittee the parameters of what, in a public hearing, in response to testimony that witnesses are giving, we as members of the committee may have a right to talk about?

I didn't raise the subject of advertising, the witness raised the subject of advertising. I do believe it is relevant in a public hearing to compare the smoking issue with the issue of alcoholism and obesity. There is some very questionable science involved here.

If the Chairman wants to focus strictly on banning smoking in Federal buildings, then why don't we just mark up the bill and move it on and not bother to have hearings. What is the point of this if we can't have a full, broad discussion on the subject?

Mr. TRAFICANT. Well, sir, you have the right to pursue any line of questioning you wish. Congress has a tendency to spin its wheels on issues not germane to the subject at hand.

Mr. EMERSON. That's very true.

Mr. TRAFICANT. I'd like to remind the gentleman that we have an expert here on respiratory diseases, not on marketing strategies for advertising.

Mr. EMERSON. Others have injected the advertising issue. I didn't say Joe Camel, he said Joe Camel. I think the Chairman is being unreasonable in trying to restrict the discussion here.

Mr. TRAFICANT. The Chairman will withdraw the concern brought to you. He wants to give you every opportunity to discuss every salient point that would, in fact, be of your concern and you have complete license.

Mr. EMERSON. I think that is appropriate as we're moving into a subject area here that is very controversial and the Chairman is my friend but I want to say to him that I don't think it's your responsibility as the Chairman to limit discussion. You may be trying to limit smoking in Federal buildings, but I don't think it's your responsibility to limit speech. I think you're being unreasonable.

Mr. TRAFICANT. I was under the impression that I was sort of trying to bring us back to the topic, not limit your speech. To clarify the record to the gentleman from Missouri, you have once again such wide latitude to proceed in any and all directions you so please, but be reminded we have specialists in here to discuss the nature of this bill. I would like to at some point get us on that.

Mr. EMERSON. I think the gentleman has a point. We have specialists who want to focus on this bill. It is my turn to be recognized and I immediately responded to the last point that Dr.

Munzer had been making and he was talking about advertising. He wasn't talking about any scientific aspect of the issue, he was talking about advertising. So I responded to that concern and you're trying to tell me it's inappropriate to talk about any issue other than what scientific expertise may be in the brains of the two witnesses at the table.

I happen to be a member of the Joint Committee on the Organization of Congress, which is trying to sort out what's wrong and what's right with Congress and we're meeting at 9:30 this morning. One of the things we need to be talking about is the conflicting committee schedules and why members who serve on more than one committee can't be at two places at the same time. We're hearing from committee chairmen today and I want to go hear what committee chairmen have to say about it.

I think this is probably the most controversial hearing I might be able to participate in today, but I think that my other responsibility at the Joint Committee on the Reorganization of Congress is over the long haul going to be more important, so I would like to submit my statement for the record, I would like to submit a legal memorandum that has been prepared in response to certain allegations that have been made about the liability concerns relating to smoking in Federal buildings.

Mr. TRAFICANT. Without objection, so ordered.

[Mr. Emerson's prepared statement and legal memorandum follows:]

STATEMENT OF REPRESENTATIVE BILL EMERSON

Mr. Chairman, I want to express my strong opposition to H.R. 881, which would ban smoking in all federal workplaces. Although there is some political sentiment to support such a measure, there clearly is insufficient science or logic to justify this extreme action.

Sound public policy decisions rely on gathering and analyzing all the evidence before the appropriate course of action is determined. However, in this case, due to the excitement being generated to over one EPA report of questionable reliability, we are considering bypassing the bulk of available evidence to arrive at a predetermined conclusion.

We have a responsibility to avoid this type of policymaking. However, in the case of public smoking, the political bandwagon is already rolling and is gathering momentum. Sadly, those on the bandwagon are ignoring the majority of scientific evidence, the rights of smokers, the realities of indoor air quality and existing policies involving workplace exposures to airborne pollutants. Although it may be politically convenient to follow this course, we would be wrongly imposing yet another burden on federal workers.

That this proposal reflects simple over-reaction to the EPA reports can be seen clearly when tobacco smoke is considered from the perspective of the many substances we encounter in daily life. Office workers are routinely exposed to a variety of so-called carcinogens in the workplace, from sources as varied as spray cleaners and tap water. And yet there is no call—from Congress, from EPA, and OSHA or from the public—to eliminate every trace of these products from the workplace. The reason is simple: at the low levels to which we typically are exposed, these so-called carcinogens are not thought to pose a risk to health.

The Occupational Safety and Health Administration (OSHA), which has responsibility over workplace safety, has studied concentrations of carcinogens to determine reasonable exposure limits—or levels below which there is no harm. Under these guidelines, it is acceptable to be exposed to a variety of substances which EPA considers to be carcinogenic, such as chromium, asbestos or benzene, as long as the amounts are not excessive.

For instance, according to OSHA, five million fibers of airborne asbestos in an average office building is perfectly acceptable. Therefore, the notion that even one molecule of tobacco smoke in the air is cause for panic is absurd and absolutely inconsistent with existing workplace policies and regulatory practices.

Even if you disregard the fact that the EPA environmental tobacco smoke (ETS) study is scientifically indefensible, the reality is that ETS is not even a major factor in indoor air quality. I am confident that when this committee takes a comprehensive and objective look at this issue, we will conclude that concerns about workplace smoking are greatly exaggerated and that a sweeping smoking prohibition is absolutely not required.

When this Subcommittee heard testimony on this proposal last month, the Chairman expressed concern that a smoking ban in federal building was needed to eliminate liability the federal government might have to nonsmoking employees if smoking continued to be permitted.

As a result of the Chairman's interest, a legal analysis has been prepared by the law firm of Covington & Burling to address that issue. I ask that this document be made part of the record.

The primary conclusion of this analysis is that a ban on smoking in federal buildings is not warranted by liability or other concerns. Specifically, the document notes that the claims made in the EPA report are not new, and so the report in and of itself does not expose the federal government to new liability.

In fact, the regulations that currently govern smoking in federal facilities already reasonably accommodate nonsmoking federal employees. Even setting aside any legal considerations, the federal government can and does accommodate nonsmoking workers. Under regulations in effect since 1987, smoking is already prohibited in most general office space in General Services Administration-controlled facilities.

The current regulations are already effective in eliminating the working conditions that, in the past, might have required nonsmoking employees to share their immediate workplace on a sustained basis with co-workers who smoke.

The federal government does not appear to risk future liability by continuing to allow smoking only in tightly restricted, separate sections. H.R. 881 is not warranted based on concerns about the government's liability.

LEGAL MEMORANDUM—A BAN ON SMOKING IN FEDERAL BUILDINGS IS NOT REQUIRED TO ADDRESS LIABILITY CONCERNS

Representative Traficant has introduced a bill (H.R. 881) to ban smoking in all federal buildings. He has suggested that such a ban is required to eliminate the liability that the federal government otherwise may have to nonsmoking employees should smoking continue to be permitted in federal buildings. In this connection, Representative Traficant has cited the recent report of the Environmental Protection Agency classifying environmental tobacco smoke ("ETS") as a Group A ("known human") carcinogen.

For the reasons discussed in this memorandum, a ban on smoking in federal buildings is not warranted by liability or other concerns. The regulations that currently govern smoking in facilities controlled by the General Services Administration reasonably accommodate nonsmoking federal employees and effectively eliminate the working conditions that gave rise in the past to claims by nonsmoking employees. H.R. 881 is unreasonable and should be rejected.

SUMMARY

H.R. 881 goes further than necessary to accommodate nonsmoking federal employees.

The EPA Report does not expose the federal government to new liability for allowing smoking in the workplace.

EPA's assertion that ETS can cause lung cancer is not new, and prior suggestions of harm have not produced an avalanche of claims against employers.

In and of itself, EPA's classification of a substance as a Group A carcinogen does not represent a determination that the substance is necessarily hazardous at levels typically encountered.

Studies of workplace smoking, which EPA ignored in its report, overwhelmingly fail to support claims of an increased risk of lung cancer.

The U.S. Occupational Safety and Health Administration should be allowed to complete its consideration of whether workplace exposure to ETS should be limited for health reasons.

The EPA Report is not legally determinative on the question of whether ETS can cause lung cancer.

Even after the EPA Report, individual claimants face nearly insurmountable problems of providing causation.

Current federal smoking regulations protect against claims under the Federal Employees' Compensation Act.

The federal government does not face potential liability under the Federal Tort Claims Act.

The Americans with Disabilities Act and the Rehabilitation Act of 1973 do not require the federal government or other employers to ban smoking.

Banning smoking would do little to address the risk of claims based on poor indoor air quality.

DISCUSSION

I. H.R. 881 goes further than necessary to accommodate nonsmoking federal employees

Wholly aside from any legal considerations, the federal government should accommodate nonsmoking employees. It should not force its employees to work under conditions that may prompt complaints of physical irritation or aggravate pre-existing conditions. The very few cases in which nonsmoking federal employees have been awarded compensation because of co-worker smoking involved situations in which the federal government has failed to follow a policy of reasonable accommodation.

The era that gave rise to such cases—an era in which nonsmoking federal employees sometimes were required to share their immediate workspace on a sustained basis with smoking employees—is largely behind us. Under General Services Administration regulations in effect since 1987, smoking is prohibited in most general office space in GSA-controlled buildings and facilities. Smoking areas may be designated in such office space only if strict conditions are met to accommodate nonsmoking employees. 41 C.F.R. § 101–20.105–3(b)(1).[1] In addition, smoking is prohibited in auditoriums, conference rooms, libraries and elevators. Id. § 101–20.105–3(b)(2), (3), (6). Smoking also is prohibited in corridors, lobbies, restrooms and stairways unless it is "not possible to designate a sufficient number of other smoking areas." Id. § 101–20.105–3(b)(4) & (c)(2)(iv). The GSA regulations are designed to serve the goals of minimizing exposure of nonsmokers to ETS while at the same time recognizing and accommodating the need of smokers. See Id. § 101–20.105(3)(a). The policy that the GSA regulations embody is one of reasonable accommodation.

H.R. 881, by contrast, rests on the premise that *no* accommodation is possible. The bill presupposes that even in the most minute quantities, and even under the most sporadic or intermittent conditions of exposure, ETS is dangerous to non-smokers, so that only by banning smoking altogether can the safety of nonsmokers be ensured and liability claims against the federal government be avoided. It is this extreme notion that the recent report by EPA is cited to support. For the reasons discussed below, however, we believe that the EPA Report does not support a ban on smoking in workplaces and that a smoking ban is not required to address liability concerns.

II. The EPA Report does not expose the federal government to new liability for allowing smoking in the workplace.

A. EPA's assertion that ETS can cause lung cancer is not new, and prior suggestions of harm have not produced an avalanche of claims against employers

The Surgeon General of the United States first suggested that ETS may be hazardous to nonsmokers more than 20 years ago, and both the Surgeon General and the National Academy of Sciences issued reports in 1986 claiming that ETS can cause lung cancer. Despite those reports (which are subject to many of the same criticisms as the EPA report), there has not been an avalanche of claims against the federal government or other employers who permit smoking.

Even Robert A. Rosner of the Smoking Policy Institute, who counsels employers on how to implement workplace smoking restrictions, has stated that he is "skeptical of claims that secondhand suits will [now] take off." Nothing that "the same

[1] Smoking areas may be designated in general office space only if the office space is "configured so as to limit the involuntary exposure of the non-smokers to secondhand smoke to be minimum; e.g., the office space must be large enough and sufficiently ventilated to provide separate smoking and non-smoking sections which protect non-smokers against involuntary exposure to smoke." Id. § 101–20.105–3(c)(2)(iii). The regulations define "general office space" as "space occupied by personnel performing their daily work functions; this includes, but is not limited to, ADP areas, mail rooms, file rooms, duplication areas, court and jury rooms, office space, etc." Id. § 101–20.105–(3)(a)(3).

basic data has been out there since the 1980s," Mr. Rosner has asked, "Why * * * should the EPA report make a difference?"[2]

To assess the true legal implications of EPA's action, it is necessary to understand what the agency's classification of ETS as a Group A carcinogen means—and does not mean.

1. In and of itself, EPA's classification of a substance as a Group A carcinogen does not represent a determination that the substance is necessarily hazardous at levels typically encountered

EPA, based on its review of studies conducted by others, has concluded that ETS is capable of causing cancer in humans—at some levels, under some conditions. In and of itself, the classification of ETS as a Group A carcinogen does not reflect any conclusion about the potency of the substance or the extent of the hazard, if any, that may be presented in particular settings.

According to Bruce Ames, a University of California scientist who developed a key test for the carcinogenic potential of substances, practically everything we eat and drink contains carcinogens, many of them naturally occurring. As a recent front-page article in the Wall Street Journal points out, even common beach sand has been labeled as carcinogenic.[3]

In fact, Dr. Devra Lee Davis of the National Research Council of the Natonal Academy of Sciences recently noted that the relative risk of cancer from chlorinated water is greater than that attributed by EPA to ETS. As Dr. Davis stated last November, at the Disinfection Byproducts Technical Workshop of the Center for Environmental Dispute Resolution in Washington, D.C., in reference to the risk of cancer from chlorinated water:

"The relative risk [for chlorinated water] is higher than the relative risk for environmental tobacco smoke. The difference is that nobody likes environmental tobacco smoke. It's kind of easy * * * for people to say, 'Oh, let's get rid of that smoke; it's really nasty and horrible,' but in fact, the relative risk we are talking about here in [a study of chlorinated water and cancer] was higher than the relative risk, for the average, for lung cancer for someone married to a smoker. Think about that."

Similarly, Dr. Morton Lippman, chairman of the Science Advisory Board committee that reviewed the EPA report, has cautioned against overinterpreting the significance of EPA's classification of ETS. When asked by a reporter about the extent of the danger posed by ETS, Dr. Lippman responded that the questioner probably had incurred a greater risk driving through Washington traffic to ask his question than he would incur in a lifetime's exposure to ETS.[4]

In this regard, it is instructive to consider other substances that EPA has classified as Group A ("known human") carcinogens:

Benzene is emitted from any form of combustion such as barbecue grills and gas-fired stoves and heating. It also is found in many office cleaning agents and in ordinary tap water. Nickel is commonly used in kitchen utensils and tableware—and even can be found in mother's milk. Chromium occurs naturally in the earth and is found in all tap water. Millions of pounds are released into the air from industrial processes each year. All are classified as Group A carcinogens but none is considered sufficiently hazardous to be banned in public settings.

The situation is similar in the case of substances that EPA has classified as Group B ("probable human") carcinogens:

Benzo(a)pyrene is found in roast coffee and coffee powders, in hamburgers, in the fumes from wood fires and barbecues, and in water and practically all air. Formaldehyde is present in most wood products and can be released from office furnishings. Aflatoxins are found in most foods.

The State of California has concluded that more than 300 substances commonly found in the environment can cause cancer. But the State has not banned them on that basis.

The U.S. Occupational Safety and Health Administration (OSHA), which is responsible for regulating exposure to carcinogens in the workplace, establishes maximum permissible exposure levels based on significant risk determinations. Typically, these limitations are expressed in terms not of a ban but of a permissible exposure limit over an eight-hour working period. For example, permissible occupational exposure limits have been set for arsenic, asbestos, benzene, chromium, coke oven emissions, nickel and vinyl chloride, all of which are classified by EPA as Group A carcinogens.

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[2] National Law J., March 1, 1993, p. 12.

[3] "Cancer Scare: How Sand on a Beach Came to Be Defined As Human Carcinogen," Wall St. J., March 22, 1993, at Al.

[4] Washington Times, April 19, 1991.

2. Studies of workplace smoking, which EPA ignored in its report, overwhelmingly fail to support claims of an increased risk of lung cancer

In classifying ETS as a Group A carcinogen, EPA considered the results of epidemiologic studies that have accumulated in the last ten years assessing reported ETS exposure in the home. Whether the conclusions drawn by EPA from those studies, or the methods it employed to reach those conclusions, are valid—and many say they are not[5]—what is significant from a legal standpoint is that EPA simply ignored available studies of workplace smoking. Of the 15 such studies that have been conducted, 13 report no statistically significant increase in overall lung cancer risk.

Both the National Academy of Sciences and Meridian Research, an independent research organization commissioned by OSHA to study the possible health effects of workplace exposure to ETS, have indicated (in 1986 and 1988 reports) that the differences in home and workplace environments make extrapolation from one to the other inappropriate. OSHA itself has emphasized that epidemiologic studies of spousal exposure in residential settings (the studies considered by EPA) are of "limited" applicability in assessing risk "under actual, prevailing occupational exposure conditions."[6]

3. OSHA should be allowed to complete its consideration of whether workplace exposure to ETS should be limited for health reasons

EPA has no regulatory authority over indoor air or ETS. So far as workplace smoking is concerned, regulatory authority resides with OSHA. OSHA may not regulate any substance in the workplace in the absence of data showing a significant risk of harm to human health based on actual occupational exposure conditions.

Prior to the release of EPA's report, OSHA consistently had rejected requests to ban or restrict workplace smoking. On January 14, 1993, after EPA released its report, the Secretary of Labor directed OSHA to commence a rulemaking addressing workplace exposure to ETS. Noting that EPA's review had been limited to studies of exposure to ETS in the home, the Secretary of Labor directed OSHA to determine the relevance, if any, of that evidence to workplace exposure to ETS.

The failure of the workplace studies to demonstrate an overall increased lung cancer risk from ETS exposure is a significant fact that OSHA must take into account. It would be precipitous for Congress to ban smoking in federal buildings without awaiting OSHA's expert determinations in this regard.

B. The EPA report is not legally determinative on the question of whether ETS can cause lung cancer

The EPA report is not legally determinative in court on the issue of whether ETS can cause lung cancer. It has no more judicial significance than the reports by the Surgeon General and the National Academy of Sciences, which also have expressed the view, based on many of the same studies reviewed by EPA, that ETS is capable of causing cancer. As noted, these reports have not produced an avalanche of claims.

Moreover, even if the EPA report were to be considered as evidence in a court of law, it seems unlikely that the report itself would ever be treated as establishing that ETS was the cause of any individual's lung cancer. Among other things, EPA's estimated "relative risk" of lung cancer in nonsmokers exposed to ETS is so low as to invite considerable judicial skepticism with regard to such a claim.

"Relative risk" compares the incidence of a disease among a population exposed to a substance to the incidence of the disease among a population not exposed to the substance. Because of the difficulty of controlling for bias and confounding in epidemiologic studies, relative risk estimates below 2.0 (1.0 signifies no difference in risk) generally are regarded as weak and either uninterpretable or unimportant.

EPA's estimated relative risk of lung cancer in nonsmokers reportedly exposed to ETS in homes was only 1.19. Courts have been quite skeptical of efforts to attach importance to such low estimates. See, e.g., DeLuca v. Merrell Dow Pharmaceuticals, Inc., 911 F.2d 941 (3d Cir. 1990) (where a plaintiff relies solely on epidemiologic proof to establish that a product caused her condition, "relative risk" must exceed 2.0 if plaintiff is to avoid summary judgment).

C. Even after the EPA report, individual claimants face nearly insurmountable problems of proving causation

Even if EPA's report were viewed by a court as establishing that ETS can cause lung cancer in humans, that still would leave unsatisfied the other prerequisites to a successful claim. In any particular case, a nonsmoking federal employee would

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[5] See, e.g. "Is EPA Blowing Its Own Smoke: How much Science Is Behind Tobacco Finding?", Investor's Business Daily, Jan. 28, 1993.

[6] Brief for the Secretary of Labor, p. 13, ASH v. OSHA, No. 89–1656 (D.C. Cir. April 5, 1991).

have to prove both that his or her lung cancer was caused by exposure to ETS (not something else) and that his or her exposure to ETS in the federal workplace (not other places) was the proximate cause of the illness. Even after the EPA report, the plaintiff would bear the burden of proof on both points.

Many factors have been associated with lung cancer in nonsmokers. Establishing that any one of them played a causative role in any particular case would be problematic at best. That would be true for factors, such as exposure to radon and diet, for which there is a reasonably strong association with lung cancer. It would be even more true for the many reported factors, including exposure to ETS, having only a weak or highly equivocal association with lung cancer.

As noted, the claimant in such a case also would have to establish that exposure to ETS at the workplace was directly responsible for his or her condition. That burden would involve many additional problems, including overcoming the fact that the vast majority of substances in ETS are ubiquitous—that is, exist at measurable levels in most environments (workplace and non-workplace) whether or not smoking is permitted.[7]

D. Current federal smoking regulations provide ample protection from claims under the Federal Employees' Compensation Act

The Federal Employees' Compensation Act ("FECA"), 5 U.S.C. 8101 et seq., provides a comprehensive remedy for federal employees who suffer work-related injury sustained in the performance of their duties. "Injury" is defined to include, in addition to injury by accident, a disease "proximately caused by the employment." 5 U.S.C. 8101(5), 8102(a). As the Employment Compensation Appeals Board has stated, "[an] injury that has some connection with the employment but that does not arise out of the employment is not covered."[8] The Appeals Board has emphasized the heavy burden on a plaintiff claiming that workplace exposure to ETS or other workplace conditions has caused disease under FECA:

"An award may not be based on surmise, conjecture, speculation, or appellant's belief of [a] causal relationship. A person who claims benefits under the Federal Employees' Liability Act has the burden of establishing the essential elements of her claim. Appellant must establish that she sustained an injury in the performance of duty and that her disability resulted from such injury. As part of this burden, a claimant must present rationalized medical opinion evidence, based on a complete factual and medical background, showing [a] causal relationship. The mere manifestation of a condition during a period of employment does not raise an inference of [a] causal relationship between the condition and the employment. Neither the fact that the condition became apparent during a period of employment nor appellant's belief that the employment caused or aggravated her condition is sufficient to establish [a] causal relationship."[9]

On several occasions, the Compensation Appeals Board has found that, although workplace exposure to ETS was not shown to have caused an employee's medical condition, such exposure did temporarily aggravate the condition. But these were atypical cases in which the federal government had failed to follow a policy of reasonable accommodation. The employees in these cases were found to have preexisting medical conditions that were aggravated temporarily when the employees were forced to share their immediate workspace with smoking employees in poorly ventilated offices. The employees were found to be unable to work under such conditions and were awarded compensation for the period during which these working conditions rendered them disabled.[10]

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[7] See generally Guerin, Jenkins & Thomkins, The Chemistry of Environmental Tobacco Smoke: Composition and Measurement (1992).

[8] In re Martinez, No. 88–937 (E.C.A.B. 1988) (emphasis added) (physician stated that none of employee's conditions was "caused exclusively" by employment-related factors including workplace exposure to ETS). A number of state courts have held that, because ETS is not an intrinsic aspect of any particular employment but is something to which people generally are exposed in myriad venues, a disease assertedly resulting from ETS exposure on the job does not "arise out of" the employment so as to make it compensable under the state workers' compensation statute. Palmer v. Del Webb's High Sierra, 838 P.2d 435, 437 (Nev. 1992); Kellogg v. Mayfield, 595 N.E.2d 465, 466 (Ohio App. 1991); ATE Fixture Fab v. Wagner, 559 So.2d 635 (Fla. App. 1990); Mack v. County of Rockland, 530 N.Y.S.2d 98, 99 (N.Y. App. 1988).

[9] In re Welke, No. 89–866 (E.C.A.B. 1989) (emphasis added and citations omitted). This standard is expressed in regulations implementing FECA, 20 C.F.R. § 10.110 (1992) (burden of proof).

[10] See In re Varela, No. 89–1189 (E.C.A.B. 1989) (employee had been forced to work in a poorly ventilated room amidst heavy smoking 8½ hours a day); In re Soo, No 89–19 (E.C.A.B. 1989) (nonsmoking employee who alleged exposure to ETS, perfume and "other chemicals" in a poorly ventilated workspace); In re Valenza, No. 88–1216 (E.C.A. B. 1988) (employee had been required to work in a "very smoky" environment and was exposed to "heavy concentrations of cigarette and cigar smoke" at work); In re Hembree, No. 82–154 (E.C.A.B. 1982) (employee who pre-

The current federal smoking regulations, as discussed, now prohibit such working conditions. The regulations thus appear to provide ample protection from such claims in the future. The lesson of these cases is that the federal government avoids claims by nonsmoking employees by following the policy of reasonable accommodation that its own regulations now prescribe.

These same cases, however, also illustrate the formidable obstacles facing an employee claiming that exposure to ETS in the workplace caused lung cancer. In none of these cases, as noted, did the Compensation Appeals Board find that the employee had established that workplace exposure to ETS had caused the underlying condition. 5 U.S.C. 8101(5).[11]

E. The federal government does not face potential liability under the Federal Tort Claims Act

Maintaining the current federal smoking policy, which allows agency heads to accommodate both nonsmoking and smoking employees, does not subject the federal government to liability under the Federal Tort Claims Act (FTCA). If the injury is one for which FECA provides coverage, the remedy provided by FECA is exclusive and the claim may not be pursued under FTCA. 5 U.S.C. § 8116(c). See Carroll v. Tennessee Valley Authority, 697 F. Supp. 508, 511 (D.D.C. 1988) (employee claiming that her lung disease was caused by exposure to ETS in the workplace was barred from asserting her claim under FTCA); Richardson v. United States, 336 F.2d 265, 266 n.1 (9th Cir. 1964) (FECA is the exclusive remedy for employee claiming injury from exposure to toxic chemicals due to employer's allegedly negligent failure to maintain a safe workplace).

Even if the FECA did not bar an employee from pursuing a claim under the FTCA, we believe that the federal government would not be subject to suit for permitting smoking in federal buildings. The FTCA bars:

"Any claim based upon an act or omission of an employee of the Government, exercising due care, in the execution of a statute or regulation, * * *, or based upon the exercise or performance or the failure to exercise or perform a discretionary function or duty." 28 U.S.C. 2680(a).

Whether viewed as involving "the execution of a * * * regulation" issued by the GSA, or as involving the exercise of discretion in the furtherance of government policy, in our view an agency head's designation of smoking areas in federal buildings pursuant to the GSA rules or like authority could not serve as a basis of liability under the FTCA. See generally United States v. Gaubert, 111 S. Ct. 1267, 1274–75 (1991) (discussing the elements of the discretionary-function exception).

F. The Americans with Disabilities Act and the Rehabilitation Act of 1973 do not require the federal government or other employers to ban smoking

The Americans with Disabilities Act ("ADA") prohibits employment discrimination by the Legislative Branch. See 42 U.S.C. 12112(a); 12209.[12] The Rehabilitation Act of 1973 prohibits such discrimination by Executive Branch agencies. See 29 U.S.C. 791(b). These statutes require the federal government, in matters of employment, to make "reasonable accommodations" to known disabilities of employees unless the accommodation would impose an "undue hardship" on the employer.

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viously had occupied a nonsmoking semi-private office was reassigned to a workspace shared with smoking employees); In re Meyer, No. 80–1719 (E.C.A.B. 1980) (employee had been required to share a workspace with smoking employees). See also Carroll v. Tennessee Valley Authority, 697 F. Supp. 508 (D.D.C. 1988) (employee forced to work in small, poorly ventilated office with coworkers who smoked); In re Marshbanks, No. 87–426 (E.C.A.B. 1987) (employee was exposed daily to "dust, cigarette smoke, stress and physical strain from lifting mail sacks"); Parodi v. Merit Systems Protection Board, 702 F.2d 743, 749 (9th Cir. 1982) (employee found to be medically precluded from working in a room in which "many other" employees smoked).

[11] In re Varela, supra note 10 (employee failed to establish that workplace exposure to ETS had caused her condition); In re Valenza, supra note 10 (employee failed to establish that his asthma was caused by workplace exposure to ETS); In re Marshbanks, supra note 10 (workplace exposure to ETS had aggravated employee's preexisting condition temporarily but condition was not caused by factors of his employment including exposure to ETS). See also In re Welke, supra note 9 (employee "has not established that her [condition] was causally related to factors in her federal employment" including exposure to ETS); In re Martinez, No. 88–937 (E.C.A.B. 1988) (employee had failed to establish that her respiratory condition and stress were caused by factors of her employment including workplace exposure to ETS); In re Spangler, No. 87–1950 (E.C.A.B. 1987) ("no medical evidence that [employee's] sensitivity [to ETS] was caused or worsened by her exposure at work").

[12] Although it is covered by the ADA, the Legislative Branch is governed by different enforcement procedures established by Congress for its employees. 42 U.S.C. 12209; EEOC's Technical Assistance Manual, Vol. 1 (Jan. 26, 1992), reprinted in Americans with Disabilities Act Manual (BNA) 90:0504.

In promulgating rules implementing the ADA, the Equal Employment Opportunity Commission made clear that sensitivity to environmental agents such as ETS does not automatically constitute a "disability" under ADA. 56 Fed. Reg. 35544, 35549 (July 26, 1991) (rules for public accommodations).[13] Moreover, even if an individual were to establish that sensitivity to ETS were a disability within the meaning of the statutory term, an official of the EEOC has emphasized that "the ADA does not require employers to have a smoke-free environment or prevent it. It does not interfere one way or the other."[14]

Indeed, a federal court of appeals has held specifically that providing an individual with a "totally smoke-free environment" goes beyond the government's obligation to provide "reasonable accommodation" within the meaning of the Rehabilitation Act. Pletten v. Merit Systems Protection Board, Nos. 88–1467, 89–1086 (6th Cir. 1990) (unpublished disposition), cert. denied, 111 S. Ct. 768 (1991). See Vickers v. Veterans Administration, 549 F. Supp. 85 (W.D. Wash. 1982) (assigning hypersensitive nonsmoking employee to work in room without smoking employees was a "reasonable accommodation" under the Rehabilitation Act).

Assuming that an individual's hypersensitivity to ETS were to be viewed as a "disability" under the ADA or the Rehabilitation Act in a particular case, it seems likely that the provision of smoking and nonsmoking areas in federal buildings as prescribed by the current federal smoking regulations would be viewed as a "reasonable accommodation" within the meaning of those statutes.

G. Banning smoking would do little to address the risk of claims based on poor indoor air quality

Tobacco smoke frequently is claimed to contribute to poor indoor air quality because it typically is the most visible component of the indoor environment. Because it is visible, tobacco smoke is the proverbial "canary in the coal mine." If employees or other building occupants are complaining about tobacco smoke, a broader problem involving inadequate ventilation may well be present. Banning smoking will not solve that problem and will not eliminate potential liability for it, especially if employees are led to believe that the employer has "solved" indoor air quality problems by banning smoking.

CONCLUSION

Legislation banning smoking in federal workplaces would be premature while OSHA is studying the matter. The federal government should accommodate its smoking employees while at the same time respecting the sensitivities of its nonsmoking employees. Reasonable accommodation is the key to avoiding claims by nonsmoking employees. H.R. 881 goes further than necessary to accommodate nonsmoking federal employees. It should be rejected.

Mr. EMERSON. Thank you, Mr. Chairman.

Mr. TRAFICANT. Is the gentleman from Missouri agreeing then that the Chairman's request to convene hearings at 8 a.m. so that members wouldn't have conflict would be agreed to on behalf of the gentleman from Missouri?

Mr. EMERSON. I think it's a wonderful idea, but I'll tell you this. I'll agree with that but I want to say this to you, you're going to run into strong opposition from about 50 members of Congress if you do it on Thursday mornings because the House prayer breakfast meets at 8 a.m. on Thursday mornings and there is a standing rule which has been violated today, there was a Democratic Caucus at 8:30 a.m. and the Republicans are doing something at 8:30 a.m., but the House prayer breakfast met at 8:00 a.m. and you were deeply missed, Mr. Chairman. Sonny Montgomery is going to be talking to you because we have an informal rule that no meeting should be scheduled at 8:00 a.m. on Thursday mornings. If you

[13] Cf. Byrne v. Board of Education, 979 F.2d 560, 565 (7th Cir. 1992) (plaintiff claiming respiratory disability failed to show that her sensitivity to aspergillus fumigatus, a common fungus found in many environments, rendered her disabled under the Rehabilitation Act).

[14] National Law J., March 1, 1993, p. 12.

want to do it Mondays, Tuesdays, Wednesdays and Fridays, that's fine, but don't do it on Thursday.

Ms. NORTON. Amen.

Mr. TRAFICANT. The gentleman from Compton, California.

Mr. TUCKER. Thank you very much, Mr. Chairman.

Inasmuch as I'm just getting here and not hearing much of the Doctor's testimony but having perused a bit of it, I'll reserve some questions at this time.

However, I do want to compliment the Chair again for bringing this matter to this committee. Obviously, this is a question of not so much the rights of smokers, but a question of the right to exist in a smoking free environment.

I would hope this committee will be able to find some type of compromise in those countervailing rights inasmuch as obviously we talk about limiting rights here and the distinguished gentleman on my left talked about his right for speech and rights for other things, that we will keep in mind that we are not here to limit the right of smokers to smoke or, for that matter, to smoke in public. I think the Chair has been more than accurate in pointing out that as much as possible, we should try to stick to the gravamen of this particular bill, that being whether or not there is going to be a smoke-free environment in public buildings as it relates to this particular legislation.

Hopefully, we will find some kind of redress and some reasonable response to that request and at the same time consider the rights of smokers and the rights of Americans who obviously have rights.

Mr. TRAFICANT. I thank the gentleman. As the gentleman from Missouri leaves early, we say adieu to you.

I'll yield to the gentleman from Tennessee.

Mr. DUNCAN. Dr. Munzer, I thought Ms. Norton made some outstanding points a while ago. Dr. Hoyt, I don't know whether you heard the tail end of his testimony, expressed some reluctance about raising the taxes dramatically on cigarettes because that's something that has concerned me.

I read an article I think in the Wall Street Journal one time that said 95 percent of the people who smoke now come from the lower income groups. We would really be placing a heavy tax on lower income people if we raise these significantly. Even those of us who don't like smoking don't feel that smokers are evil people. It seems to me that we should focus our efforts on trying to help smokers rather than punish them.

From that, what I'm getting at is I thought she made an excellent point when she said you do an excellent job on the back end but what about the front end. What is the American Lung Association doing? Are you spending any money at all on helping people overcome the addiction or are all your efforts focused on publicity and that type of thing?

Dr. MUNZER. One of our major programs and the one that we are most proud of is called, "Freedom from Smoking." It's a smoking cessation program that is made available in communities around the country. It's a program constantly being updated to make sure that it reflects the latest in addiction science. It's available in a variety of self-help materials, programs for the workplace, videos that

people can use in their homes. We are even now looking at interactive computer programs.

So we are doing a lot to try to help people kick the smoking habit. I am including information regarding the Association's Freedom from Smoking program and other smoking cessation efforts we are conducting. (See page 450.)

Mr. DUNCAN. Are those programs working? Do you think those things are working and what percentage of your budget are you spending on things like this? Is this a minor part of your activities?

Dr. MUNZER. No, it's not a minor part. It's a substantial part. I don't know the specific percentage that's involved in these programs because we are a very large and varied organization. I would like to submit, along with my testimony, a copy of our organization's annual report which highlights expenditures for our activities. (See page 485.)

Mr. DUNCAN. This is what I'm saying. To summarize, I agree with Ms. Norton. I think maybe you ought to increase your efforts on that end rather than on the other end. I don't think there's anybody in the country that doesn't know that smoking is not a healthy thing, but it's like Mr. Emerson said, sometimes I think I'm addicted to ice cream. I know I eat way too much ice cream and it's bad for me but what I'm saying is, people know it's bad for them, I think, almost everybody, but yet, as you said, it is as tough to overcome this as it is to overcome an addiction to heroin and cocaine. So we need to do more.

Dr. MUNZER. It's as addictive. That doesn't mean that it's impossible for people to quit smoking because the vast majority of people who quit smoking do so entirely on their own, in spite of all the factors that promote smoking. That's the good part of the story. An increasing number of Americans, indeed, are quitting and an increasing number of Americans are concerned about the residual effects of environmental tobacco smoke, and that's what brings us here.

Mr. DUNCAN. Thank you very much.

Mr. TRAFICANT. I want to thank these two distinguished witnesses for coming in. Again, under the unanimous consent we have, you will be available for any written questions and we'd ask you reply and respond. It need not be extensive writing, basically germane and basic to the question at hand.

Thank you both for coming and participating in a process that I am sure will be quite unusual.

Thank you very much.

Our next panel of two witnesses consists of Mr. Gray Robertson, President, Healthy Buildings International, Fairfax, Virginia and the second witness is Dr. Gio Batta Gori, Director, Health Policy Center, Bethesda, Maryland.

I'd ask again as you get started, please do not read your extensive statements. They will be included in the record. Please summarize so we can interact with you and have time for such activity.

I'd start with Dr. Gio Batta Gori.

TESTIMONY OF DR. GIO BATTA GORI, DIRECTOR, HEALTH POLICY CENTER; AND GRAY ROBERTSON, PRESIDENT, HEALTHY BUILDINGS INTERNATIONAL

Mr. GORI. Thank you, Mr. Chairman.

I'm Gio Batta Gori. If you remember, I was here at the last hearing on H.R. 881 on March 11th. The Tobacco Institute asked me to be present here again to clarify some of the issues raised in the testimony submitted by Dr. Munzer on March 11th in writing. As usual, I express my own views and not necessarily those of the Institute.

Dr. Munzer claims that two large studies, the Stockwell and the Brounson studies published after the release of the EPA report support the EPA conclusions about ETS. As you will see, the studies support rather the opposite conclusion.

In my written submission to you, I have included copies of the data of the Brounson and Stockwell studies and I urge you to take a look at them. They are not that difficult to understand. Even lay people could make sense of what is written there.

According to the Stockwell data, risk of lung cancer appears increased if the information about ETS exposure comes from the woman patients or from their husbands. However, lung cancer risk is much decreased if the exposure information is provided by siblings and relatives, suggesting therefore that ETS exposure may actually decrease cancer risk.

This raises the obvious question of who gives correct information about ETS exposure. Is it the women themselves with lung cancer who may be induced to find blame for their illness in someone else's smoke, or is it possibly the less-biased siblings and relatives. Could husbands, for instance, give reliable information about their spouse's exposure to ETS during childhood, as the Stockwell data reported? Could such data lead to reasonable conclusions other than the results are utterly confusing. Are the authors justified in concluding that ETS exposure "increases the lung cancer risk in women who have never smoked"?

In contrast to the Stockwell data, the Brounson data for childhood exposures, for instance, show a consistently decreased risk of lung cancer, as much as one-half the risk of people not exposed to ETS.

I don't want to imply here that childhood exposure to ETS means protection, but the data surely beg the question and cast reciprocal doubt on the opposite results obtained by Stockwell.

The Brounson studies for adults exposed to ETS show that except for the very high cigarette reports, ETS exposures are consistently associated with reduced risk for lung cancer. Should one conclude that ETS exposure actually protects these people?

The women exposed to ETS from the husband alone, which is the only index of exposure that EPA has considered in its reports, show no elevated risk of lung cancer as a group. Actually, the group including ex-smokers shows a reduced risk of ETS exposure. Again, should one conclude that ETS protects them?

This set of results clearly conflicts with the overall conclusions of the authors and also with the EPA report, yet incredibly Brounson and Stockwell alike write that the results are consistent with an increased risk of lung cancer from passive smoking.

Brounson and colleagues also stated smoking restrictions in workplaces are well-advised even though their own study, the Stockwell studies and most other studies, report no risk association for worksite exposures to ETS.

There is also the important question of statistical significance in the EPA report. While it's true that statistical significance is a matter of consensus, it's also true that universal consensus prescribes a margin of error of 5 percent or lower and frowns upon the 10 percent margin that EPA insists using in the ETS report, In fact, EPA would certainly reject the petition for registration of, say, a pesticide if it were submitted to the agency with data based on a 10 percent statistical standard.

Professor Dockery, who I believe will testify today, and whom Dr. Munzer quotes, is right when he states that the statistical significance of data, "reflects the stability, that is the uncertainty of the effect estimate." Indeed, by adopting a 10 percent instead of a 5 percent standard, the EPA report aims at giving an artificial impression of stability and precision in the data. By this gambit, the data are not changed but their intrinsic uncertainty is fictitiously reduced.

The Stockwell and Brounson studies are the latest among the best of available ETS studies, all of which suffer from similar incurable contradictions? Polling their results by META-analysis, as the EPA has done, does not remedy the inherent flaws. Rotten apples don't make good applesauce.

The reason for this confusion is that ETS exposure over a lifetime cannot be defined with any credibility by anyone, and the effect of other lung cancer risk factors is not accounted for, as I've indicated in my previous testimony. It is only by starting with a preconceived assertion that EPA can reach its conclusions and only by selecting data that sustains this preconception and by disregarding systematically all contrary evidence.

My purpose here, Mr. Chairman, is not to cast aspersions on the ETS studies, but rather to illustrate how contradictory and essentially uninterpretable the results are if analyzed comprehensively in a true weight of evidence exercise. In fact, the weight of evidence of which the EPA report speaks is nothing but an exercise in selective use of data and, therefore, not a weight of evidence at all.

Mr. Chairman, members of the committee, the real issue in front of you, I believe, is not passive smoking, as you may think, but rather, the credibility of a regulatory agency. If EPA wishes to claim that its policies are based in science, then it should follow those rules of scientific evidence that any scientific institution or individual scientist is expected to observe. Indeed, it is strange that at a time when scientific misconduct is hotly debated in our midst and is the burning focus of many congressional hearings, official agencies of our government are praised and are proud to operate under scientific pretensions that would bring swift condemnation to any outside scientist or institution.

This, Mr. Chairman, concludes my remarks and I'm ready for your questions.

Mr. TRAFICANT. Thank you, Mr. Robertson?

We'll come back with questions as a panel.

Mr. ROBERTSON. Good morning.

Thank you, Mr. Chairman and members of the committee for the opportunity to speak to you today.

I'm following up on the presentation of Mr. Turner. Mr. Turner, an employee of mine, deputized for me in your last hearing. I know that he drew your attention to the fact that we specialize in studying indoor pollution problem issues in major buildings. We've investigated over 1,000 major buildings, housing over a million people around the world.

I'd also point out that whenever I am asked to address such groups as the American Institute of Architects or the Association of Energy Engineers or ASHRAE, the Gas Council and many of the other distinguished associations, we get nothing but compliments on the willingness we have to share our research and expertise.

However, if I'm asked to give precisely the same presentation at the request of the tobacco industry, I'm branded as an apologist to that industry. I'm not. Our company consists of a team of consultants who believe in speaking factually on what our findings are. We are a very practical-oriented company looking for solutions to problems.

We are dealing today with a problem of environmental tobacco smoke in buildings. I'd like to first point out that one of the prior speakers, Dr. Munzer, made the statement that he's concerned, his Association is concerned, with the escalation of indoor air quality issues throughout this country. He also points out that in studies that we've had done by Lou Harris Associates, the public opinion group, less than 18 percent of 25,000 people questioned in this country said that they are exposed to tobacco smoke in their offices.

Indeed, in many, many buildings we go to today, either there are complete smoking restrictions or in some cases, bans. Yet, in precisely those same buildings, we are finding some of the sickest present in this country. The fact is that we ourselves strive to improve the air quality in any building and that would certainly include trying to ensure that nonsmokers are not exposed to excessive amounts of tobacco smoke.

There is a limit to how far one can go in protecting smokers and nonsmokers from any type of indoor pollutant. Certainly the outside so-called fresh air throughout this country today contains many compounds that also are classified by the Environmental Protection Agency as Group A carcinogens. If you are using this air to bring into a building and dilute pollutants in a building, there is a limit about how far you can go. You cannot obtain the unobtainable. You cannot produce an environment which is totally free of all these different pollutants.

The question is, how do you manage it? On the one hand, you can follow the route that most if not all the hospitals follow, as pointed out by Dr. Hoyt, you can introduce a total ban. He already addressed the fact that this creates at least one known problem, the problem of litter and the unsightliness of so many people standing outside the building smoking.

Many companies approach us and say, how do we handle our problem because the above is not the sole problem with a total ban. Other problems occur. The most important one is loss of productivity. Too many people, senior executives, key technical people, are not at their desks when needed. They are outside smoking.

Secondly, there is a public image problem. It looks bad when you approach companies and see many smokers outside the building. In fact, one major building we were dealing with, a foreign visitor at the time presumed they had a picket line outside the building and he passed comment that this was very bad for the public image.

Also, I'd point out that if you'd look at, for example, an airport where a total ban has been introduced, say Dallas-Ft. Worth, you have a situation where the smokers outside the building have to be negotiated by visitors to the airport. Naturally, they would be exposed to tobacco smoke during the negotiation through these smokers.

Contrast that with Salt Lake City Airport in Utah where they have designated smoking lounges that work very, very well indeed. People who want to go into the lounge can do so. So if you're a non-smoker, you need never be exposed to that particular tobacco smoke.

That, in fact, is the theme of my presentation. You can offer solutions to the problem inside major buildings. We would suggest that every time I approach a major client who has asked us to give them help in suggesting smoking policy options, we follow a menu approach. The start of it is, first of all, do you have the correct ventilation requirements in your building to deal with all indoor pollutants, including tobacco smoke? Basically, are you operating to the latest ASHRAE ventilation standards; do you have the right filtration standards?

In our experience, if you do, almost invariably you have no problem in those buildings with tobacco smoke. This is the status quo as far as a well-ventilated building is concerned. If we meet those conditions, we say fine.

Also, we can prove that if you meet those ventilating standards, even the return air containing residues of tobacco smoke, once it goes back to the main ventilating unit, the air handling unit, once it's filtered and diluted with outside air, the resultant supply of air to the building meets all the national primary ambient outside air quality standards defined by the Environmental Protection Agency. So if you've met those standards, how can you possibly opt to do better? You're already delivering air back to the building that meets outdoor requirements.

Our first suggestion is, if you get your ventilation and filtration standards correct, you can accommodate a moderate amount of smoking.

I understand for social reasons, political reasons or whatever, you might wish to go further than that. As a result, we would certainly work with our clients and design designated smoking areas. The first step is the possibility of introducing a designated smoking area without physical barriers. You see this often done very successfully. In fact, Salt Lake City Airport is a case in point.

If you ensure that the smoking area has sufficient exhaust ventilating capacity, you create a negative pressure in the zone where the smokers are. Water cannot flow uphill; air cannot flow against a pressure gradient. As a result, if you exhaust the smoking area correctly, with its own dedicated exhaust ventilation capacity, you do not even need a physical barrier to separate the smokers from

the nonsmokers and you have no movement of smoke from the smoking area to the nonsmoking area.

So my first option would be the status quo with ASHRAE ventilation standards. The second is a designated smoking area without physical barriers and the third is logical, if you now want to make it even more certain, use physical barriers by all means. We design designated smoking lounges and ensure that those lounges are fitted with adequate ventilating capacity, mainly exhaust air capacity.

We've even gone as far as designing lounges with the most sophisticated ventilating systems available in the world today, using the principles of displacement ventilation. It's a very simple principle. We move the air so that it comes from a clean source and moves past the occupants to a source of exhaust and there is no recycling of that air. It's easy to retrofit in existing buildings. We are using, and I describe in my testimony, the techniques that Halton, the Finish company, for example, use very successfully in retrofitting existing rooms to turn them into rooms with displacement ventilation.

I would point out that even smoking lounges, when fitted with correct displacement ventilating techniques, can provide air quality superior to many operating rooms that I've examined in this country. So it can be done and it can be done practically and economically.

I think the main theme that I would like to point out is the fact that tobacco smoke itself is an object lesson. We've described it in the past as a litmus test. To illustrate this point, let me return for a moment to the example I cited of the operating room.

In this country today, the ventilation in operating rooms is obviously of paramount importance. You do not want to have problems with cross-infections, you do not want to have problems with all the anesthetic gases present in the room. One easy check to ensure how efficient an operating room is working is to release some synthetic smoke in the room and watch to see which air currents conduct the smoke away and you time it to see how expeditiously it takes it away.

If the smoke does not move, then you can rest assured that the bacteria and fungi will also be trapped and so will the anesthetic gases with disastrous results. Use precisely the same analogy when you go into a building. If you go into an office and see a large volume of smoke, you're seeing visible evidence of deficiencies in ventilation.

All too often in the past we've taken the quick, easy way out and hang up a no smoking sign, but we've not addressed the root cause of what trapped that smoke in the first instance. If the smoke was trapped, all the indoor pollutants present must also be trapped and they will come and haunt you later on. The best example of that is the Environmental Protection Agency's headquarters building where several years ago, they were so concerned with the levels of tobacco smoke in the building that they introduced an effective ban in most areas and very severe restrictions in others.

Then in an essentially smoke-free building, several months after the fact, so many people were getting sick in the smoke-free building that they started to picket the building and now you have a

number of those people who have filed a $39 million lawsuit against the managers of the building for being exposed to indoor pollutants in what was proven to be an inadequately ventilated building.

In fact, when you think of it, plaintiffs' attorneys will be very quick to seize on the fact that if you've taken a quick way out and simply hang up a no smoking sign, you are admitting that your ventilating system cannot adequately manage an adequate quantity of tobacco smoke. If it can't, by definition, it cannot be meeting the ASHRAE ventilating standards, so subsequently if you've got people sick in your building, you're admitting liability of having kept them in a badly-ventilated building. I think that should be a major concern.

So our theme, constantly is, by all means let's work to reduce the levels of tobacco smoke in all buildings, but lets work at it in a sensible and progressive way. We've been suggesting, and our testimony gives specifics, on how to operate a "building systems approach" which is very basic and very simple. All the technology exists today and it's simply following recommended ventilation rates, filtration standards, inspection standards, standards for commissioning the building properly, and correct standards for operation and maintenance. If you do that, your buildings can indeed handle all indoor pollutants as well as the tobacco smoke. Of course if you want to then arrange separate designated smoking areas, it is very easy to accomplish that.

I think the main theme facing this committee is that if you determine that the regulatory action is needed to control indoor air quality, then the approach shouldn't be by source control, it should be by a pragmatic, effective and nononerous series of steps to adopt a building systems approach.

Remember the EPA points out that indoor pollution in this country costs between $10 and $12 billion a year in lost productivity. I'd also point out that several studies have shown that respiratory tract infections in this country account for $15 billion in medical care costs and there's hard evidence to suggest that 50 percent of that, $7.5 billion, is caused by indoor pollution inside buildings.

So I think the message is simple. If you want a technique when the government is currently seeking cost-cutting methods and you're concerned with medical health care controls, can there really be a better option that has no association with any pork barrel politics? A building systems approach is a positive, stated goal of improving this country's competitiveness, I think this is an absolute win-win situation for everyone—for government, for the managers, and for the employees.

Thank you very much, indeed. I will answer any questions on my testimony.

Mr. TRAFICANT. Mr. Robertson, do you agree then that smoke, ETS trapped in a building without adequate ventilation is dangerous to your health?

Mr. ROBERTSON. I think in any inadequately-ventilated building, you have many dangers to your health—bacteria, fungi, dust, et cetera. In our studies, smoke has been low on the list of what the major concerns have been, but I would agree that any time you

allow an accumulation of any indoor pollutant, including smoke, it's not very sensible.

Mr. TRAFICANT. Let me see if I can revisit that. Let's see if I can focus in on your testimony.

Are you telling this committee that the concentration of ETS trapped in inadequately-filtered buildings is dangerous to one's health?

Mr. ROBERTSON. I am saying that if it's allowed to accumulate to high concentrations, it can adversely affect and can impact on peoples' health.

Mr. TRAFICANT. Fine. In addition to your testimony, you've stated that there are also a number of other pollutants. These other pollutants in an inadequately-ventilated, filtrated system are dangerous to your health. Is that a correct statement?

Mr. ROBERTSON. Correct.

Mr. TRAFICANT. What specific studies do you have cited by specific scientific resource units of a credible nature that have documented any other specific indoor pollutant of significant quantity to have reached at this point a contrast or to be a comparison with the tremendous volume and reams of studies that have singled out, to start with, major contributors of indoor pollution, that being ETS smoke? Would you comment on what specific pollutant and what study and what has been brought forward from the scientific community?

Mr. ROBERTSON. There's many, many occasions when the scientific community itself accepts that all indoor pollutants have created major problems. I, before the National Academy of Sciences, have already made presentations revealing all our findings and documenting the numbers of cases when we've found things like fungus or bacteria to have been a major source of any particular problem. These have always been in far greater numbers than have been the case when we look at the incidences where tobacco smoke has been a problem.

Mr. TRAFICANT. Let me revisit my question. We have reams and reams of documents and scientific study supported by what is considered to be, and what has been testified by professionals, as contributing to this scientific data, to be of a credible nature that ETS, passive, secondary smoke, is an indoor pollutant that will kill and contributes to a tremendous health problem in America.

On line with that, and considering ETS in a milieu of indoor pollutants as you've discussed them, what other specific indoor pollutants have ever been cited for specific studies which can be brought forward in a case of law, in a worker's compensation case, for example, with adequate data to support one other specific indoor pollutant to be so studied?

Mr. ROBERTSON. Workman's compensation cases in Florida have implicated aspergillus fungus, of causing the adverse health effects of the people and it was judged to be so. I can point out there's been many other cases where bacteria and fungi have made people sick, and I would point out that there's far more successful lawsuits throughout this world implicating other indoor pollutants as causing sickness in buildings than has ever come to trial yet for environmental tobacco smoke.

Mr. TRAFICANT. How many of these other indoor pollutants you cite have been formally investigated, studied, and formal reports concerning their contributory damages to our society have been published?

Mr. ROBERTSON. Many of them. There is far more documented reports on asbestos, on radon, on formaldehyde, on benzene.

Mr. TRAFICANT. What is the conclusion of any one of those studies that have discussed radon, benzene or asbestos?

Mr. ROBERTSON. In every single case, there's been a move by the large government agency with the responsibility of managing such substances in the environment, OSHA, to set permissible exposure limits for those particular substances. Those permissible exposure limits are set at such a level that if you keep the concentrations below those, every worker can be exposed to them without fear of adverse health effects. The same thing will inevitably have to come in once OSHA starts making regulations about what the acceptable level will be for environmental tobacco smoke.

Mr. TRAFICANT. To the best of your knowledge as a building process specialist—I might add, are you a scientist?

Mr. ROBERTSON. I'm a chemist.

Mr. TRAFICANT. Being that your field is dealing with healthy buildings, is it not a fact that the government makes specific amounts of awards and grants to remove asbestos and other types of known indoor pollutant problems from the workplace in the environment where possible?

Mr. ROBERTSON. They have made grants certainly. They also give advice in other areas not to touch it. In many cases, the EPA has recently revised their own decisions and opinions that in many cases, it would have been better to leave well enough alone and leave the asbestos in the buildings without disturbing it. Far more people have been exposed to asbestos as a result of tearing it from the buildings than ever would have been had it been left in place.

That is not to say that there aren't some buildings that we ourselves make recommendations that the level of asbestos is so bad that they must deal with it as promptly as they can.

Mr. TRAFICANT. Does Healthy Buildings have a smoking policy?

Mr. ROBERTSON. We do not have a smoking policy. We have discretionary smoking. Also, we have good ventilation.

Mr. TRAFICANT. The GSA has informed the Congress that it would cost anywhere between $90 to $130 million to retrofit our Federal space to adequately filtrate, ventilate secondhand smoke.

Mr. ROBERTSON. If they are saying that, what they really are saying is that is the amount of money that you should be spending to protect the health of the workers in your buildings and avoid having them exposed to indoor pollutants period, not any specific indoor pollutant. I think that would be a very smart investment to make if that's what they can accomplish.

Mr. TRAFICANT. The gentleman from Tennessee.

Mr. DUNCAN. Thank you, Mr. Chairman.

Mr. Robertson, just to kind of sum up, assuming that all of us want to make our buildings as healthy as possible, what you're saying is that the singlemost important thing we can do is to try to make sure that our buildings are adequately ventilated?

Mr. ROBERTSON. That's correct, sir.

Mr. DUNCAN. Your saying that there are many other extremely hazardous substances that are in the air that EPA has identified as being Class A carcinogens besides environmental tobacco smoke? That's one of the factors, but you said it ranked low.

Mr. ROBERTSON. I'd like to illustrate the fact that if you look at a known Class A carcinogen, for example, benzene, every single published study in the United States on concentrations of benzene has been reviewed and published in peer-reviewed literature. I'm looked at that literature and in the materials I've submitted to you, there is one particular table that points out that if one examines the concentration of benzene in the air of a nonsmoking home, you have higher concentrations than you do in a smoking lounge, not just the discretionary smoking office, a dedicated smoking lounge. That ought to be of concern if the benzene concentrations were significant. The fact is, they are not, in either case. The permissible exposure limit for benzene is 1 part per million. The concentration of benzene in all the smoking offices that have been studied throughout this country is 1,000 times less than that.

I totally agree with OSHA that it's at such an insignificant level that why should we be concerned. I think that's the key issue that we're dealing with.

Mr. DUNCAN. I'm not the expert that you are on studying buildings and so forth, and you've studied over 1,000 buildings?

Mr. ROBERTSON. That's correct.

Mr. DUNCAN. I assume you would say that every building is different and that some buildings are well-ventilated and some aren't?

Mr. ROBERTSON. Correct.

Mr. DUNCAN. Are you saying you think that the best policy for us would be to make sure that our buildings are all well-ventilated, but are you saying also that we should ban smoking except in designated smoking areas?

Mr. ROBERTSON. I would say the most important thing to recognize is that in all our studies, over 80 percent of the problems we've come across in buildings is because building engineers are operating the buildings the wrong way. They are making decisions, usually motivated to try and reduce energy costs. They are shutting the outside air intakes on buildings and recycling the air inside the building.

I can't tell you how often that is just the wrong decision. They are increasing the energy costs many times when they do that. Once you educate them, they listen to us and follow our recommendations.

Let me try and make this a very simple explanation. The average temperature when you're supplying air conditioned air into a modern building today, you would pitch the supply temperature at 55 degrees. That's what you're going to deliver into an office to cool the people. The heat from the people, the heat from the lights, the heat from the equipment has to be removed, so you use the cool air to mix with the air in the room. The room average may be 70 degrees Fahrenheit. The supply temperature is 55 degrees Fahrenheit.

So if you were to recycle all the air in the building, you would be having the room air at 70 degrees going back to the air conditioning unit and has to be cooled to 55 degrees before it goes back

into the room. The person shuts the outside air intake saying he's saving energy and if the outside air happens to be say 50 degrees on that day, it would be much cheaper to direct all the room air in the building outside the building and bring in 100 percent outside air.

I said if it happened to be 50 degrees. Let's assume it was only 30 degrees. If you mix 50 percent air at 30 degrees with 50 percent air at 70 degrees, you get air at 50 degrees, you do not need any cooling. So you can have 50 percent outside air come into the building with no cooling costs. Indeed, anytime the temperature of the outside air ranges from about 30 degrees right up to 70 degrees, it is cheaper to bring more outside air into the building than recycle air in the building.

I've been to so many hearings, I've been to so many associations of engineers, et cetera, and heard repeatedly, and I know the National Institute of Occupational Safety and Health repeatedly points out, that building engineers have made decision themselves to save energy and reduced the outside ventilation rates. They've shut the outside air intakes in buildings. They need to be educated. In many, many cases, they are going the complete opposite way. They are putting up the energy costs, and worse still, they are trapping all the pollutants in the building and contributing to the sickness of these people.

By increasing the ventilation, you can actually reduce indoor pollution, you save money and you get better environment for long periods of time throughout this country. I fully appreciate the extremes of temperature when it is necessary to bring in limited amounts of outside air, mix it with return air, et cetera, but the key is that over great periods of time by boosting ventilation rates, you reduce energy costs and you certainly improve health. That's an educational step that this country needs.

Mr. TRAFICANT. Will the gentleman yield on that?

Mr. DUNCAN. Yes, sir.

Mr. TRAFICANT. That would imply then, Mr. Robertson, that in the extreme when the temperatures are rather low, you'd be talking about having two systems in place in the building?

Mr. ROBERTSON. No, sir. The same heating and cooling capacity exists, you just don't use it when you bring in boosted amounts of outside air. That, by the way is defined as an economizer cycle and most buildings have the capability to operate an economizer cycle.

When you go to the extremes, you still mix the small amounts of extreme outside air, whether it's hot or cold, with the buildings return air, your existing equipment can easily accommodate the heating or the cooling requirement.

Mr. TRAFICANT. Thank you.

We'll come back to the distinguished gentleman from Tennessee.

Mr. Tucker?

Mr. TUCKER. Thank you very much for your testimony, Mr. Robertson. It's been extremely lucid.

You remarked at the statistic projection by GSA of $90 to $130 million for retrofitting Federal buildings as a figure that would be well worth spending and perhaps very reasonable in order to retrofit the buildings for adequate ventilation.

Mr. ROBERTSON. I said if it handled indoor pollution in total.

Mr. TUCKER. Of course that gets right back to your prior testimony just a minute ago that that kind of ventilation system would save on productivity and it would save on the problems of indoor pollutants overall?

Mr. ROBERTSON. That is correct. I think I should qualify it. I've not seen the specific details of that estimate, so I'm not really making an engineering judgment that it's the right amount. I'm saying the principle is correct. If you spend a substantial amount and achieve that goal of dealing with all indoor pollution, then you will get massive returns and improved productivity, reduced absenteeism and reduced medical costs in this country.

Mr. TUCKER. In your studies, you said you obviously had assessed about 1,000 buildings. Were these buildings included in the private sector, public sector or both?

Mr. ROBERTSON. Both. We do a significant number of buildings for the Government, we do many, many buildings for the major property developers throughout this country, and we do a lot of work for the major Fortune 500 companies. Finally, there would be schools and universities included as well, plus a significant number of hospitals.

Mr. TUCKER. Have you ever assessed any buildings on Capitol Hill?

Mr. ROBERTSON. Yes, we have. We've worked for the Architect of the Capitol and we studied the Longworth Congressional Building next door.

Mr. TUCKER. So your findings have been consistent with those buildings too?

Mr. ROBERTSON. Absolutely.

Mr. TUCKER. I certainly appreciate your testimony. It tends to speak of the kind of sensitivity to both sides of the issue that I think anyone who is concerned about both sides of the issue would find reasonable.

I appreciated your stories about the airports, Dallas-Ft. Worth as opposed to Salt Lake City Airport and how you can have a kind of flip side effect.

Mr. ROBERTSON. What astonishes me in that case, I spoke with the architect of Salt Lake City Airport, we happened on that by happenstance. One of my executives was traveling back home from a vacation and he stopped at Salt Lake City Airport and he noticed the smoking lounges—it was in a Delta terminal—and was so impressed with them. They had no doors on them and yet there was no movement outside, all the air was streaming in.

He called and asked could he speak with the architect who subsequently spoke to me. He said that they get nothing but compliments on their design and that other airports are looking to follow them. It just shows a very down-to-earth, practical approach that can be used. Isn't that preferable to the situation that has just occurred very recently here in Virginia where a major hospital has come to us and said, we've got a problem, we're sick and tired of losing doctors, nurses and staff disappearing outside into the grounds to smoke. It looks bad, when you approach, of all things, a major hospital, and see people huddling outside doors and entrances smoking. Is there a way they can accommodate them? Can we design designated areas for them that if they want to, they can

go to, people know where they are, they can improve their productivity, and reduce the unsightliness. They are paying us to make those recommendations.

Mr. TUCKER. Apparently, in the case of the Dallas-Ft. Worth Airport, what you seem to be implying is that not only was there a case of loss of productivity but the nonsmokers' right to be free from the ETS smoke was also being infringed upon as they entered the airport?

Mr. ROBERTSON. Exactly. Doesn't it make a lot more sense if the people were in properly designated areas that they have the freedom of choice not to go into those areas?

Mr. TUCKER. It does if you're trying to accomplish the goal of protecting the rights of the nonsmoker.

Thank you. Nothing further at this time.

Mr. TRAFICANT. Mr. Clyburn?

Mr. CLYBURN. Mr. Chairman, thank you very much.

Let me ask permission that my prepared statement be entered into the record.

Mr. TRAFICANT. Without objection, so ordered.

[Mr. Clyburn's prepared statement follows:]

STATEMENT OF HON. JAMES E. CLYBURN

Mr. Chairman, and Members of this Subcommittee, I appreciate these hearings and the opportunity being accorded me to address this Subcommittee. The issue before us is one which is highly charged to say the least. Yet on the other hand, representing a Congressional District which depends in large measure upon the legal crop necessary for the production of cigarettes causes me great pause. Tobacco products are responsible for over 15,000 jobs in my area. About 42 percent of the 6th Congressional District depends upon tobacco and its related industries as the primary sources of family incomes. Over 8,000 of these jobs are held by undereducated farm workers whose lack of educational opportunities is due in large measure to issues and policies far beyond their control. They are hard working people. Many of them, in spite of their own lack of educational opportunities have made significant sacrifices to educate their own children and grandchildren. I feel compelled to advocate fair treatment for the industry and the affected families, many of whom have little hope for a decent living otherwise.

I understand the emotions involved here and have great concern for them. I would hope however that a resolution can be found which not only protects the health and welfare of those who do not wish to have their space violated by cigarette smoke, but also protect the rights of those, who because of their individual choices, do use tobacco.

I am proud of the work that the chairman and this subcommittee are doing in this area and I am confident that a viable and reasonably accepted resolution can be found.

Mr. CLYBURN. Thank you so much.

Since I was not here to hear all of the testimony, I'm not going to ask any direct questions. Let me say this though. From what I gather to be the spirit of that testimony, and knowing my friend Mr. Tucker's attitude on this subject, I get a good feeling that maybe we're getting to the point where there can be some reasonable accommodation for everyone involved in this.

Thank you so much, and I'll listen to the rest of the testimony.

Mr. TRAFICANT. I appreciate it, Mr. Clyburn.

I have a few more questions for Mr. Robertson and then I have a few for Dr. Gori.

Mr. Robertson, these other indoor pollutants, are they voluntarily brought into the building?

Mr. ROBERTSON. I think you can argue that some of them are. If you choose one particular type of carpet over another and you know the one you choose happens to have a higher emission rate than the other, then you're voluntarily bringing it into the building, yes.

Mr. TRAFICANT. Then it would be reasonable to assume, with all of the litigation—minded, new America that if it could be established perhaps in a court of law that the owners and/or those in control and custody of a particular building, knowingly brought into a building a known exposure, they could in fact at least be questioned under a liability class statute for such decision. Is that a reasonable assumption?

Mr. ROBERTSON. That is happening.

Mr. TRAFICANT. With that in mind, there is no smoke in a Federal building or any other building unless someone ignites literally a tobacco product?

Mr. ROBERTSON. There is no environmental tobacco smoke, you've still got the vast bulk of the constituents that are present in smoke in the same building.

Mr. TRAFICANT. I am focusing now on a case of liability.

Mr. ROBERTSON. Fine.

Mr. TRAFICANT. We have 7,800 buildings in the Federal pool. Most of them are not being built today, Mr. Robertson, that could be built with all these new state-of-the-art improvements that specific professionals who could be helping advise us like yourself, could have a play in and a part in. These are existing buildings with the faults that lead to tremendous costs of retrofitting and/or adjustments to accommodate our new technological gains and understanding of indoor air quality.

I get one thing from your testimony. You, in fact, make a strong case for designated smoking areas, is that correct? Is that an assumption I can make from your testimony today?

Mr. ROBERTSON. I'd like to answer the first question to address the fact about your older buildings. I have some good news for your. In many, many cases, the older buildings have far higher ventilation rates than the new modern buildings because they were designed and built before the obsession to reduce energy costs. So the good news is many of your older buildings have far higher ventilation rates and if managed properly, and if the engineers know how to operate them properly, then you can achieve ventilation rates that exceed the modern ASHRAE ventilating standards. So that's the good news.

With regard to your second question, am I supporting the use of designated smoking areas, I would say without question, if the building is poorly ventilated, yes, I would like to see designated smoking lounges and I would like to see them with exhaust ventilation capacity and we routinely recommend that to our clientele.

However, on balance, I would prefer to see the building itself operating with adequate amounts of ventilation and if that's the case, you do not need the designated smoking lounges, although I will accept that there's many cases when we would still recommend them because that's the client's wish.

Mr. TRAFICANT. Then you're saying in this release of data now to support the dangers of secondhand smoke, that Congress, in its

attempt to deal with this new scientific admission, is at least on the right track in attempting to make the workplace safer and we can assume then, from the broad parameter of your testimony, that you're saying, let the smoking be a highlight, it's brought attention, but beware and look at all indoor pollutants if there is to be a policy. Is that a reasonable assessment?

Mr. ROBERTSON. I think that's reasonable.

Dr. Gori, you believe that secondhand smoke is safe for nonsmokers in a given confined area?

Mr. GORI. What do you mean for a given, confined area, Mr. Chairman?

Mr. TRAFICANT. A reasonable confined area of existing office space both in the private and the public sectors.

Mr. GORI. I don't know that it's safe. I believe that there is no evidence that it is not safe.

Mr. TRAFICANT. Are there any positive health benefits, Dr. Gori, to those who smoke tobacco on a fairly regular basis, to the best of your professional knowledge and/or opinion?

Mr. GORI. What kind of benefits are you talking about, Mr. Chairman?

Mr. TRAFICANT. Any positive health benefits?

Mr. GORI. It depends on whether you consider health the feeling of well-being, satisfaction, and happiness and everything else.

Mr. TRAFICANT. So under the broad parameters of the question I asked, you've inserted the mental health aspects and the emotional capacity of smoke, but leaving the mental health aside, leaving the psychological aside, leaving the emotional stability aside, in talking about the human body, is there anything good about smoke for the human body in a physiological impact capacity?

Mr. GORI. There is mounting scientific evidence of some of the health benefits of smoking. Of course we have a great deal of record about the negative effects of smoking as well, but there is evidence based on the same kind of epidemiologic data that is used to show the negative effects of smoking which shows that, for instance, uterine cancer is 50 percent less frequent in women who smoke than otherwise; colon cancer seems to be also reduced in smokers, perhaps 30 percent. These are data published by the same agencies that collected data about the negative aspects of cigarette smoking.

You have probably a 50 percent reduction of prostate cancer in men. These also are data published in many studies. There are not as many studies as you would have about the negative aspects of smoking because obviously it's not very popular to publish about the positive aspects and the health benefits of smoking. The old record about the reduction of Parkinson's disease; there is a good record showing that ulcerative colitis is very much reduced, as much as half the risk in smokers as nonsmokers, and of course, osteoarthritis seems much lower in smokers than in nonsmokers. So there is a substantial record of evidence. It is not as publicized, not as well-known, of course, and it doesn't have the same amount of publications that the negative aspects of smoking have obtained.

Mr. TRAFICANT. Are we to assume then from your testimony that you're saying the focus has been on the negatives of smoking and there are many good things and positive health things about smok-

ing that perhaps in this fervor to demean the dreaded weed that we've overlooked all of the benefits to woman and mankind from tobacco?

Mr. GORI. I don't say that we're overlooking them; I say that the data are there for anybody to see if they want to see them. I don't think there is a great deal of desire to see that there is any benefit associated with smoking.

Mr. TRAFICANT. But you're certainly not saying that the tremendous number of professionals who have assessed, studied, evaluated, investigated this smoking phenomenon, both direct and secondhand, that you're not implying in any way that they are smoking something other than tobacco and don't know what they're talking about, are you?

Mr. GORI. I cannot speak for them, Mr. Chairman. I don't know what their motivations are. The only thing I can say as a scientist is that the record is weighted on one side, not necessarily on the other.

Mr. TRAFICANT. For every cause, an equal an opposite reaction, right?

Mr. GORI. No. There are probably things that have only negative effects. I don't think that anybody would find a benefit in the virus that causes influenza or in the bacillus that causes tuberculosis, so there are things that have only one side, but the smoking issue, of course, has many sides, as you can imagine.

Mr. TRAFICANT. Are you being compensated for your visit today to the committee?

Mr. GORI. Of course. I must add that my compensation represents only a small part of my annual income. I'm not receiving all my compensation from any particular agency like EPA or anybody else.

Mr. TRAFICANT. Who is compensating you for your visit here today?

Mr. GORI. I'm sorry?

Mr. TRAFICANT. Who is compensating you for your visit here today?

Mr. GORI. The Tobacco Institute.

Mr. TRAFICANT. Do they think smoking is bad for your health, Dr. Gori?

Mr. GORI. I cannot speak for the Tobacco Institute on this issue, Mr. Chairman.

Mr. TRAFICANT. I thought you just testified you were here on their behalf.

Mr. GORI. For this particular hearing which is ETS.

Mr. TRAFICANT. Mr. Robertson, you have already testified that you were here testifying on behalf of certain interests?

Mr. ROBERTSON. That's correct.

Mr. TRAFICANT. But it is your professional opinion, and were it not even for that compensation, you would not be here, is that correct, sir?

Mr. ROBERTSON. Very willing to do so.

Mr. TRAFICANT. I want to thank both of you. You've made some very strong cases that certainly will not fall on deaf ears. The subcommittee, as you can recognize from today, is not without diversity, as is the Congress, and we will attempt to promulgate some

policy rather than wait until we are swamped by the public which Congress many times does.

We appreciate your testimony. You also may be open for questions, if you will, and I ask for brief responses to us in the event that should materialize.

Thank you very much.

Our next panel will consist of Mr. David C. Zeigler, Acting Assistant Secretary, Occupational Safety and Health Administration, U.S. Department of Labor; Mr. Joseph M. Woodward, Associate Solicitor for Occupational Safety and Health, U.S. Department of Labor; and Mr. Lawrence Rogers, Director, Office of Workers' Compensation Programs, U.S. Department of Labor.

With that, I would like to open, if there would be no objection, with Mr. Rogers, Director, Office of Workers' Compensation Programs because we are very concerned about several things before we open up your testimony here, Mr. Rogers.

A court held in SHMP v. New Jersey Bell Telephone Company, that the "right of an individual to risk his own health does not include the right to jeopardize the health of those who must remain around him or her in order to properly perform the duties of their jobs."

Efforts to win workers compensation benefits for victims of passive smoke in the workplace have been successful now in at least five States. In New Mexico, a worker collapsed at work due to an allergic reaction to ETS and was later unable to get a job due to such activity and the State Appeals Court ruled in that specific case that this individual was disabled under the workers compensation law attributable to ETS.

With that, this panel was put together because Congress faces, without doubt, a liability question here in its failure to act and the sin of omission could be more dangerous than the sin of commission. This is what we want to know and you are right now at the heart of one of our concerns, in addition to the health of the American people and its workplace.

We'll start with you, Mr. Rogers.

TESTIMONY OF LAWRENCE ROGERS, DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS, DEPARTMENT OF LABOR; AND DAVID C. ZEIGLER, ACTING ASSISTANT SECRETARY, OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION, DEPARTMENT OF LABOR, ACCOMPANIED BY JOSEPH M. WOODWARD, ASSOCIATE SOLICITOR FOR OCCUPATIONAL SAFETY AND HEALTH

Mr. ROGERS. Thank you, Mr. Chairman.

I'm here today as the part of the Labor Department that handles workers' compensation for the Federal Government. The Federal Employees Compensation Act is the basic workers' compensation protection for the 3 million Federal employees worldwide, wherever they are and whatever they are doing.

I have submitted to you a statement that goes through our process and tells how it works and I'm not going to recover that ground since it's here on the record. I'd just like to go on to say that for this particular program, we would cover exposure to toxins, in this case tobacco smoke, in the workplace and, in fact, under the Fed-

eral Employees Compensation Act, we have awarded workers' comp benefits to Federal employees who have been affected by tobacco smoke in the workplace.

The circumstances for making those awards depends on the fact of the injury, that the person was there, that there was smoke in the environment, that the person was in the performance of their duty, and a medical cause or rationalization, as we call it, that shows a linkage between the condition the person had as a result of the exposure, and the medical evidence that we would rely on.

We have not, historically, kept data that would let me tell you how many cases there have been. We simply don't classify things that way. We have gone out in preparation for this hearing and asked our offices if they recall particular cases and we were able to identify 10 or 12 cases that people recall on an anecdotal basis where we have either paid or are now paying benefits.

Most of the benefits are paid as a result of aggravation of existing conditions, allergies, asthma, things of that sort. We have a few cases where the person's aggravation became permanent, that is, they did not recover from the aggravation and we have continued to pay benefits to that person over a prolonged period of time. We have not identified through this process any cases that led to death or lung cancer that we could tell you about.

As far as the liability issue and workers' compensation, the employer is protected in the sense that it's called exclusive remedy. If something happens to you in the workplace for the Federal employee, the employee's recourse is to file a workers' compensation claim and that does limit the liability of the Federal Government to that employee. The benefits would be paid to the employee or any family members through this system. There is no tort liability in this situation.

We can't tell you that we've seen an increase in the number of these cases. Certainly the growing body of evidence in medical science, I think, has made people more aware of the possibility that they are being harmed; growing sophistication in allergy identification has made it possible for doctors to make the case that their patient was affected by tobacco smoke, and I think that has led to these claims being approved.

Even if the numbers grow, I think we've seen a change in policy in our buildings, a reduction in the smoke, and in that sense, we would expect fewer. However, at this juncture, we do cover tobacco smoke. The FECA program has been in existence since 1916 and we have paid some claims and would continue to do so where the facts of the case made it evident that we should.

Mr. TRAFICANT. Thank you.

Mr. Zeigler?

Mr. ZEIGLER. Thank you, Mr. Chairman, for this opportunity to discuss H.R. 881, The Ban on Smoking in Federal Buildings Act.

The Administration generally supports your efforts to protect the health and safety of Federal workers. Clearly, H.R. 881 addresses a serious health issue, one which OSHA has under very active review at this time. The OSH Act charges OSHA with setting occupational safety and health standards when there is a significant risk of injury or material impairment of health.

In order to show significant risk, OSHA relies on research and other technical support from other agencies, such as the National Institute for Occupational Safety and Health, NIOSH, EPA, and on the private sector. Safety and health protections provided under the Act extend to private sector employees at a virtually unlimited variety of work sites.

Under Section 19 of the OSH Act, and Executive Order 12196, we have authority to provide for safeguarding of Federal employees' safety and health through a requirement that Federal agencies comply with standard-setting consistent with those issues regulated by OSHA for private sector employers.

OSHA's past efforts to address the overall issue of indoor air quality have focused on providing information and guidance to our field personnel to be used in the evaluation of indoor air conditions in the Nation's workplaces. While our past efforts to address indoor air problems have been somewhat limited, the agency recognizes the seriousness of the issue and is now going beyond these initial efforts to review the need for rulemaking to address indoor air quality and passive tobacco smoke.

In September 1991, OSHA published a Request For Information to the public in the Federal Register to obtain information on indoor air conditions, including passive tobacco smoke. OSHA is currently reviewing more than 1200 comments submitted in response to this RFI in order to determine the appropriateness and feasibility of an indoor air standard.

OSHA is also working closely with many other agencies, as I mentioned earlier, who have a role in various aspects of indoor air quality, including HHS, the Surgeon General, NIOSH, EPA, and GSA. As part of this interagency cooperation, EPA's recent report on "Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders," has been included in the public docket for OSHA's RFI on indoor air quality.

EPA's report identified passive tobacco smoke as a known human Group A carcinogen and indicated that it is responsible for approximately 3,000 excess lung cancer deaths a year in nonsmoking adults. OSHA views this as a significant study which was developed using a credible methodology, and it will be considered by the agency, along with all other comments, in order to determine further agency action.

With regard to H.R. 881, OSHA supports the objective of the bill to protect Federal employees and the public from the hazards posed by passive tobacco smoke in Federal buildings. The overall intent of the legislation is consistent with OSHA's mission to assure safe and healthful working conditions.

Mr. Chairman, we appreciate your efforts to focus public discussion on this important issue. Thank you again for the opportunity to testify. I'll be glad to answer any questions you may have.

Mr. TRAFICANT. Thank you, Mr. Zeigler.

Mr. Woodward, do you have a statement you want to make to the committee?

Mr. WOODWARD. No, I don't.

Mr. TRAFICANT. Do you have any statement to be submitted for the record?

Mr. WOODWARD. No. I'm here accompanying Mr. Zeigler.

Mr. TRAFICANT. Are you support for Mr. Zeigler? [Laughter.]

Mr. WOODWARD. Right.

Mr. TRAFICANT. Given the explosive nature of this issue, and wondering just how many people gathered in this room may smoke, but today not one person has smoked, nor has anybody smoked at any of our hearings. There's becoming a conscious awareness of scientific, medical discovery or awareness that documents health problems associated with both smoking and now for the first time, secondhand smoke, individuals who do not smoke, but are subject to debilitating disease and perhaps even fatality due to another's willful behavior.

The question, as you can see, is a fine one on rights here. Congress must deal with those rights and the emotionalism that comes out is inherent to any process that goes on in the Congress.

Now you're in here because there is another animal here. The Government is exposed to potentially huge dollar liability problems. With that in mind, H.R. 881 says this, you can smoke but you can't smoke in the building. In that regard, to safeguard the building, is the Office of Workers Compensation thoroughly in agreement with that position, Mr. Rogers?

Mr. ROGERS. Well, as Mr. Zeigler said, we're in support of the thrust of the bill. We think that a clean air environment is desirable. We have paid some claims where it was shown that people were exposed to secondary smoke.

I think, on the other hand, there has already been tremendous changes in buildings. No one smoked in this room today and certainly my memory of the Congress several years ago is that it's quite a change. In our own building, for example, we do have a nonsmoking policy with designated areas and the change from 10 years ago is amazing. I think the exposure is down significantly as a result of it.

Mr. TRAFICANT. You have designated smoking areas?

Mr. ROGERS. Yes, sir, we do.

Mr. TRAFICANT. There are an awful lot, as testified earlier, indoor pollutants that contribute within a closed or trapped system inadequately ventilated or filtrated. There are other indoor pollutants that are contributors to health problems. How many such cases has the legal community brought forth in any other indoor air quality workers compensation case that you know of?

Mr. ROGERS. I don't know if you'd call it indoor or not, but it's exposure to asbestos. There have been a lot of asbestos claims that we have handled in the system. Most of them related to exposure during the Second World War in shipbuilding, but a number of those kinds of claims have been paid.

Again, this is not a tort system, it's totally an administrative system. Claims are not reviewed by the courts. The law explicitly prohibits review by the courts, so we don't see the classic sort of court case that I think you're talking about. We have seen other things, exposure to floor cleaners that were improperly used; other chemicals; in my own memory, up to and including American Legionnaires Disease where the bacteria were present, verified, and we have paid claims like that.

Mr. TRAFICANT. In SHMP v. New Jersey Bell Telephone, the court held "The right of an individual to risk his or her own health

does not include the right to jeopardize the health of those who must remain him or her in order to properly perform the duties of their jobs." Do you agree with that ruling of the court?

Mr. ROGERS. I don't know if I can speak to the ruling, but would say that the policy that's being pursued in our own building, HHS and other places is that a judgment has been made that people shouldn't be subjected to it, either for health reasons or for aesthetic reasons, but clearly the movement in the Executive Branch has been to limit exposure to smoking. I think it's based on a judgment like that.

Mr. TRAFICANT. In your professional decision, dealing with workers compensation cases, do you believe that the finding in SHMP v. New Jersey is fairly much the standard by which this matter will be legally resolved in the future?

Mr. ROGERS. Your pinning me to a case that I just don't know much about.

Mr. TRAFICANT. The general parameters of that case are that you have a right, perhaps, but that right doesn't spill over in the workplace where you can affect the rights of others.

Mr. ROGERS. And I think that basic decision has been made in the policy in the Federal buildings. I think there are very few Federal buildings, none really that I know of, where smoking is still allowed completely in the workplace.

Mr. TRAFICANT. Mr. Clyburn?

Mr. CLYBURN. Mr. Rogers, did I hear you correctly, you said the building that you occupy has designated smoking areas?

Mr. ROGERS. Yes, sir, that's correct.

Mr. CLYBURN. What kind of building is that?

Mr. ROGERS. You can see it from here. It's the Labor Department building between 2nd and 3rd Streets on Constitution Avenue. It's a five-or six-story building, covers almost two square blocks, and is a standard office building with a lot of interior space and sealed windows with a ventilation system.

Mr. CLYBURN. Sealed windows?

Mr. ROGERS. Yes, sir.

Mr. CLYBURN. The area designated for smoking is separately ventilated?

Mr. ROGERS. The areas are a few restrooms that have been identified as having ventilation that we believe is the best that can be provided in the building and smoking is restricted there. The Department started out originally restricting smoking in the work environment and leaving smoking in the hallways. That was found to be too obtrusive and after negotiations with the collective bargaining agency, it was restricted to a few restrooms in the building. Then there is an area set aside in the cafeteria where people can smoke as well.

Mr. CLYBURN. You heard the testimony earlier from Mr. Robertson about the age of buildings and whether or not they were properly ventilated. How old is your building?

Mr. ROGERS. It's about 15 years old.

Mr. CLYBURN. Can I conclude then that you decision, or whoever made the decision, to set aside these smoking areas, you are aware of the court case that the Chairman just mentioned?

Mr. ROGERS. I don't think it's been driven as much by a specific court case as a growing concern about exposure to tobacco smoke that's based on the evidence developed by studies and the objection of a lot of people saying I don't want to be exposed to this. As a result of that, the Department, in this case, but I think the Executive Branch certainly, broadly decided to minimize that exposure.

Mr. CLYBURN. Let me clear up that question a bit. You all are aware of the possible concerns surrounding litigation?

Mr. ROGERS. As I explained, for the employer-employee relationship in the situation there would not be a tort liability.

Mr. CLYBURN. So you think what you are doing in this instance adequately addresses those liability questions?

Mr. ROGERS. I think from the Department's standpoint and the will of the people who work there, that it's the best solution at this point. If a bill were passed saying no smoking in the building at all, I think a lot of people would welcome that, but the exposure right now is very minimal at this juncture.

Mr. CLYBURN. Would all people welcome it?

Mr. ROGERS. No, I don't think so because there are some people who still smoke and who think their rights are being infringed on not to be able to smoke wherever they want. You know this.

Mr. CLYBURN. Yes, but their rights aren't being taken away, we're just modifying a bit.

Mr. ROGERS. Right. They can still smoke, just not in the building.

Mr. CLYBURN. We all have to modify our rights a bit, don't we?

Mr. ROGERS. Yes.

Mr. CLYBURN. Mr. Chairman, in the statement I've entered for the record, I feel compelled to state that I hope this is an issue which we can find some reasonable resolution to and to keep in mind that I am very concerned that peoples' rights are, in fact, protected, but I think we ought to keep in mind that we ought to allow people to make certain choices in this country. We're now seeing, I think in a broader sense, a revisit of issues such as helmets being required.

We see States now, Mr. Chairman, my State being one of them, that are very upset at the directives coming from on high that says we're going to take your Federal dollars from your highways if you don't pass this kind of law, if you don't make people wear helmets and all these kinds of things. These are problems that people are beginning to have, so I think we ought to be very, very careful how intrusive we get with Federal policies when we start talking about what States are allowed to do and what others are allowed to do in the management of their lives. So I'm very, very concerned about this. I just want us to be sure that we are careful.

Mr. TRAFICANT. I appreciate that.

Let me mention this to the gentleman. We do not deal in with this bill with any State law, nor do we impose any requirements on anyone except the Federal Government, our workplace and those particular buildings in which we have control and custody. I agree with you 100 percent but that's why the bill is narrowly crafted to avoid that.

Before I get to all of the panel, I have another question for Mr. Rogers. You have designated smoking areas at the home offices of the Office of Workers Compensation, correct?

Mr. ROGERS. Yes, sir.

Mr. TRAFICANT. Where are they?

Mr. ROGERS. They are in a series of restrooms in the building and an area in the cafeteria that has been blocked off with some planters and dividers and that sort of thing.

Mr. TRAFICANT. Adequately ventilated?

Mr. ROGERS. As best I can tell. I think if you get real close, you certainly can smell it. The cost of changing ventilation of the building is high, as you have mentioned, so the Department has tried to identify places that seem to be best ventilated and designated those as smoking areas.

Mr. TRAFICANT. So you've attempted then in your officeplace to segregate as much as possible the smokers from the nonsmokers so as to reduce the impact of ETS secondhand smoke?

Mr. ROGERS. Yes, that's correct.

Mr. TRAFICANT. Because of the cost, there has been no significant or specially-directed filtration systems?

Mr. ROGERS. That is correct.

Mr. TRAFICANT. A smoker and a nonsmoker could, under your policy, conceivably go to the potty in the same facility?

Mr. ROGERS. They could. The restrooms are designated though very clearly and the one closest to my office happens to be a smoking restroom and I make the trip way on down the hall.

Mr. TRAFICANT. But there are no specific ventilating systems for those bathrooms?

Mr. ROGERS. That's correct.

Mr. TRAFICANT. And when the door opens, that smoke could, in turn with the air pressure and air flows in our pressure patterns in our buildings, be admitted to the hallway?

Mr. ROGERS. I don't know that I could say that. You said specially-ventilated. They are not specially-ventilated, but they are ventilated and so I think that the pressure question is one in which it's not likely to seep out into the hallway.

Mr. TRAFICANT. One more question for you. The Surgeon General years ago said, red light; EPA says red light; Defense attorneys say green light; Congress says deep pocket. In your opinion, do you see an exposure factor of increasing liability magnitude with the reaffirmation and confirmation of the original Surgeon General opinion with that of the documented, fact-finding of the Environmental Protection Agency?

Mr. ROGERS. I think because of the actions that have been taken and the reduced exposure, that the liability is being offset by the knowledge that has been gained about the ill effects, frankly. That, I think, is why we don't see a larger number of cases.

If we had this knowledge 30 years ago and with the smoking patterns we had 30 years ago, I think we would see a huge number of cases, but I think action that's already been taken reduced that possibility.

Mr. TRAFICANT. To maybe clarify my question a little further, given that in 1993, there has been a confirmation of the Surgeon General's report of years ago, that in the years to come perhaps by the turn of the century, without addressing ourselves to policy, in your opinion as Director of the Workers' Compensation Programs for our Government, do you see an increased liability exposure that

could emanate from a Congress that fails to adequately address these findings with appropriate policy?

Mr. ROGERS. I just can't answer that in the affirmative because of the facts of the exposure going down. In fact, because the Executive Branch, at least, has moved to reduce the exposure, I think sort of looking at in the years ahead, if a person came in and said my claim is because of tobacco smoke, and factually, you couldn't establish that happened in the workplace, it's going to be from somewhere else and the Government would not be liable in that case.

Mr. TRAFICANT. But they'd have to establish that?

Mr. ROGERS. Yes, it would be factual to show it happened at the workplace.

Mr. TRAFICANT. Is it reasonable to assume that the possibility of that type of litigation could ensue?

Mr. ROGERS. It certainly could ensue.

Mr. TRAFICANT. Thank you.

Mr. Zeigler, you have an Executive Order, to ban smoking at the White House. If there is an Executive Order to ban smoking in all buildings, point blank, go outside, what would be the status of that on collective bargaining agreements and courts of law in a contested scenario?

Mr. ZEIGLER. It would not change our position, Mr. Chairman, in terms of our responsibilities to look at all the issues associated with indoor air quality and passive tobacco smoke. We have to weigh those issues and make our decision without regard to the Executive Order.

Mr. TRAFICANT. Isn't it a fact that an Executive Order would not apply to collective bargaining agreements, and if Congress was to adequately address the issue, it will be through Federal law? Is that a correct statement?

Mr. WOODWARD. An Executive Order certainly could so provide for collective bargaining agreements.

Mr. TRAFICANT. Given the matter at hand and with a number of buildings, 7,800, and the hundreds and hundreds of thousands of square feet of rental space, and given the tremendous data now coming forth, the Department of Labor has certainly addressed itself to these concerns, correct?

Mr. ZEIGLER. Correct.

Mr. TRAFICANT. What is the policy of the Department of Labor?

Mr. ZEIGLER. The policy at the Department of Labor, as Mr. Rogers stated, has been an evolutionary policy. A policy that started with smoking in offices and has changed and tightened a couple of times in the last 5 or 6 years. Smoking was initially prohibited in offices but allowed in hallways; more recently, as Mr. Rogers said, smoking has been prohibited in hallways and offices and limited to specific restrooms and one area of our cafeteria where there is some separate ventilation.

Mr. TRAFICANT. Has OSHA previously been involved in a rulemaking associated with safety and health and smoking?

Mr. ZEIGLER. Not as a specific issue, Mr. Chairman. We do have a regulation on asbestos. As part of that regulation, we require smoking cessation programs for asbestos workers because of the tie-in with asbestos and smoking.

Mr. TRAFICANT. What studies have led you to assume the combined synergistic effects of those combinations?

Mr. ZEIGLER. Evidence was provided to us, as part of the rulemaking process from a number of sources that convinced us that there was, in fact, a direct tie-in. That was part of the rulemaking process notice and comment from the public, public comments considered by the agency, and ultimately the rule issued.

Mr. TRAFICANT. How about explaining a term for me—exclusive remedy as it relates to Federal compensation claims? Anyone?

Mr. ROGERS. Let me do that. Workers' compensation is a social contract and what happened when workers' compensation was established is that basically what had been settled in a tort environment was handled administratively. The tradeoff was that the employee no longer could sue the employer, but they were supposed to get a quick, no-fault kind of decision on their workers' compensation claims which before had often dragged on for many years in courts. The sort of descriptor of that is exclusive remedy.

The employee's exclusive remedy for recourse where injury occurs in the workplace is to go to the workers' compensation system for medical costs or for wage loss replacement. That's exclusive remedy and that's why I say you don't see this in a tort situation in the employer-employee relationship.

Mr. TRAFICANT. Mr. Zeigler, did the staff at the Department of Labor analyze the provisions of the Americans Disabilities Act as they pertain to an otherwise qualified, disabled individual who is prevented from taking a job due to smoking in the workplace?

Mr. ZEIGLER. That would be, Mr. Chairman, part of our analysis during the overall rulemaking process, as I explained earlier. It's an issue that we would look at and consider if the agency determines that such a rulemaking is appropriate and possible.

Mr. TRAFICANT. The gentleman from North Carolina, Mr. Valentine?

Mr. VALENTINE. Mr. Chairman, I thank you very much.

I came here to hear the testimony principally of Dr. Dockery and so I will have questions for him. I am very tempted to pose some of those questions to these gentlemen, but will, for the sake of time, not do so.

Thank you, Mr. Chairman.

Mr. TRAFICANT. I thank the gentleman for helping me in a previous committee with other jurisdictional business here earlier today. Thank you.

Mr. VALENTINE. Always a pleasure to do business with you, Mr. Chairman.

Mr. TRAFICANT. We had several members that had to leave here on other business who would like you to remain open for questions that could be submitted from the members. Your answers could be brief and right to the point, we would hope, and the record will be open for your answers.

Are there any other questions? Mr. Clyburn?

Mr. CLYBURN. Yes, Mr. Chairman. I have two questions.

I appreciated your inference to the red light and the green light. There is one other light, Mr. Chairman, that you skipped over, and I hope that the Congress will recognize the amber light. It may be a light for us to pay a little attention to.

I want to ask a question about the employees who do have the right to bring the workers' comp charges. This may go to what I heard Mr. Zeigler say, but either one of you may answer.

If an employee were to bring a workers comp claim because of smoking in the workplace, would that employee carry the burden of having to prove that whatever ailment that smoking is alleged to cause did not occur in the home or in some other place of recreation? What happens to the employee who brings a claim against the Labor Department or any other governmental agency if that employee's spouse may smoke in the home?

Mr. ROGERS. The answer to the burden question is that the employee does have the burden at the outset to prove the allegation.

Mr. CLYBURN. You mean that the smoking problem took place at work and not at home?

Mr. ROGERS. Whatever the claim is. The Department of Labor is actually the adjudicator for the entire Federal Government in this particular workers comp system and wherever the employee is, whoever it is, and whatever agency, the employees have the burden of proving that what they say happened to them happened to them—it had to be at work, had to be a part of their duties, and there has to be a causal relationship.

I like to say that because someone had a heart attack at work doesn't mean work caused the heart attack. So when an individual case is filed with us, we look at all of those factors. Mr. Zeigler mentioned smoking and asbestos. Almost all the asbestos cases had smoking with it and you had to sort those out.

We depend a great deal on the adequacy and the rationality of the medical evidence that the doctors bring to bear in an individual case and the facts behind it. If people are exposed to smoke outside the workplace, and there is no history or factual evidence that there was smoke in the workplace, then this is not a claim that would make it.

Mr. CLYBURN. That was the other part of my question, Mr. Zeigler. In those claims where you had dual claims coming forth, did I understand you to say that you all made the determination that there was a causal relationship?

Mr. ROGERS. There has to be a causal relationship. Smoke in the workplace would have to cause the injury that the person suffered.

Mr. CLYBURN. I understand that. I'm not asking that question. I'm talking about this connection between smoking and asbestos. I'm trying to get at what Mr. Zeigler was saying. You're saying that you all have determined that there is some connection?

Mr. WOODWARD. Part of the evidence in the rulemaking record for OSHA's asbestos standard showed that a person who smokes has worse effects from exposure to asbestos or a higher degree of risk from exposure to asbestos than a person who does not smoke, and for that reason, OSHA has put in a requirement that employers, where there are high levels of exposure to asbestos, should offer smoking cessation programs. It was based on some evidence of what's called a synergistic relationship between asbestos exposure and smoking in bringing about adverse health effects.

Mr. CLYBURN. I certainly would like to see some of that. I've heard a lot about it and knowing something about some of the tort

cases in asbestos, this is the first I've ever heard that tobacco had something to do with that. This is mind boggling to me.

Mr. TRAFICANT. Will the gentleman yield? Perhaps if there are any findings or studies or paperwork or documentation you have of the synergistic effects of combining asbestos and tobacco smoke, that you would make them available to our subcommittee? Would that be possible? You do have those types of studies?

Mr. ZEIGLER. In fact, it was born out of the process in a court order, so we'd be glad to provide that to the committee.

Mr. TRAFICANT. Without objection, so ordered.

[The following was received from Mr. Zeigler:]

Attached are two studies concerning the synergistic effect between asbestos exposure and tobacco smoke submitted to the OSHA public comment docket (Docket #H–033C) for the November 4, 1983, Occupational Exposure to Asbestos Emergency Temporary Standard (FR vol. 48, p. 51085). The Selikoff study is Exhibit 84.190 and the Hammond study is Exhibit 84.47 in the asbestos docket. These studies were cited in the preamble to the 1986 final rule on asbestos (51FR at 22625).

MORTALITY EFFECTS OF CIGARETTE SMOKING AMONG AMOSITE ASBESTOS FACTORY WORKERS[1, 2]

(By Irving J. Selikoff, M.D.,[3] Herbert Seidman, M.B.A.,[4] and E. Cuyler Hammond, Sc.D.[4, 5])

ABSTRACT—Nine hundred and thirty-three amosite asbestos factory workers began work from June 1941 through December 1945. At 20 years from onset of employment (1961–65). 582 were known to be alive and to have had asbestos work experience solely at the factory. Smoking habits were ascertained at 20 years from onset of employment, and observation was then maintained prospectively through 1977; 304 deaths occurred, approximately twice the number expected. The excess mortality was largely due to malignant neoplasms; 116 neoplasms as compared to 33 expected. Sixty cancer deaths were from lung cancer and 14 more were due to mesothelioma. Nevertheless had it not been for cigarette smoking, many of the excess deaths would have been avoided. Asbestos alone would have accounted for much of the increase, including some excess deaths from lung cancer, but not for most of the deaths (60) from this neoplasm. With the use of smoking associated mortality rates, it was calculated that the combination of cigarette smoking and asbestos exposure increased the risk of lung cancer death about 60 times. Thus the experience of the amosite asbestos factory workers agreed with that of insulation workers and provided evidence that the asbestos-smoking interaction is specific for asbestos rather than other possible influences in industrial environments.—JNCI 65: 507–513, 1980.

Abbreviations used: ACS = American Cancer Society; BE = best evidence available; DC = death certificate information only.

It was shown in 1967 that asbestos exposure sharply increased the already high risk of death from lung cancer associated with cigarette smoking among asbestos insulation workers (1). The limited data on which these observations were based were later extended and confirmed by much larger studies, which demonstrated that between 1 in 4 and 1 in 5 deaths among these workers was due to lung cancer if they smoked cigarettes. In addition, as compared to other nonsmokers, nonsmoking insulation workers also had an increased risk of lung cancer, but inasmuch as risk among nonsmokers has been relatively low, even multiplying this risk severalfold

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[5] We thank Mr. Ashley Bodden, Mr. David Newman, Mr. Edwin Silverberg and Ms. Marie Carbo for their assistance in processing the data; Ms. Dorothy Perron, Ms. Shirley Levine, Ms. Rivla Margoles, and Mr. Charles V. Nolan for the field investigations of the men in this study; Ms. Frances Perez for management of specimens and X-rays; and Ms. Selma Annenberg, Mr. Sidney Sibel and Ms. Judy Marmor for preparation of the manuscript. We acknowledge the help of the pathologists, clinicians, hospitals, coroners, State health departments, and other medical facilities providing detailed information, records, slides, blocks, and roentgenograms. We also thank Dr. J. Churg, Dr. Y. Suzuki, and Dr. M. Kamerstern for their help.

did not result in many lung cancers (2, 3). Cancer of the esophagus was also considerably increased among smoking insulation workers but not among their nonsmoking co-workers. However, pleural mesothelioma, peritoneal mesothelioma, cancer of the stomach, and cancer of the colon and rectum occurred with approximately equal excess among both smokers and nonsmokers (3).

These studies were detailed and prolonged because we preferred to utilize only prospective observations made after establishing the smoking history from the individual concerned, with subsequent observation, and to compare observed deaths with those expected among men of the same age, in the same years, with the same distribution of smoking habits, also observed prospectively. This situation led to some delay in accumulating data to compare with our original observations, but we believe that this method has made the comparisons more secure.

We have been conscious of the fact that our findings have been derived from one category of asbestos-exposed individuals—asbestos insulation workers. Whereas it has been clearly established that these individuals have very significantly increased risks of death from asbestos-associated diseases (4, 5), there remained the possibility that other factors in their work environment could have interacted with their cigarette smoking exposure and that some or all of their increased lung cancer risk could be so attributed. It has therefore been of interest to us to investigate the mortality experience of another group of asbestos-exposed workers who did no insulation work and who were not employed in the construction industry.

We now report results from this study. We found that factory workers employed in a plant making amosite asbestos products for shipyards and other industrial facilities has lung cancer mortality experience consistent with that of the insulation workers. Asbestos exposure alone increased the risk of lung cancer in the absence of cigarette smoking, but for reasons given, the total number of such cancers was not great. On the other hand, it appears that the remarkable increase in risk among cigarette-smoking asbestos workers is due to the exposure to the mineral dust rather than other factors, multiplying the already very high risk of cigarette smoking.

MATERIALS AND METHODS

In 1941, with the encouragement of the U.S. Navy, a factory was established in Paterson, New Jersey, to manufacture asbestos products for the armed forces and for industrial uses. We have ascertained that amosite asbestos was used almost exclusively (with very small amounts of chrysotile asbestos also being used). Our analyses of fiber retrieved from the factory's storerooms, filters from workers' dust masks, samples of the products made, and fibers extracted from workers' lungs at autopsy have confirmed this almost exclusive use of amosite asbestos and are consistent with the fact that the product specification called for such fiber, that the machinery was designed specifically for this purpose, and that the company that supplied the asbestos from its South African mine had provided only this fiber variety (6). This specificity is of some interest because it provides information concerning one of the three major fiber types. Insulation workers had been exposed only to chrysotile asbestos at first and later to both chrysotile and amosite (5).

In 1941–45, 933 men began work in this plant. Some worked for a month or less and others until the plant closed in 1954. We have sought to trace the entire cohort of 933 men and to keep them under surveillance. We have already found that this group has, overall, a significantly increased risk of death from asbestos-associated disease (7).

Other studies have shown that the risk of death from neoplasms known to be associated with asbestos exposure becomes considerable only after 20 years from onset of exposure to asbestos. We therefore believed it useful to limit our analyses to the experience of the men who worked in this plant, starting with their 20th year from onset of employment and excluding all experience subsequent to asbestos employment elsewhere. For some men (those who began work in 1941) the experience was from 1961 onward. For others, observation for current purposes did not begin until 1965 (i.e., men who began work in 1945). Of the original cohort of 933 men, 351 did not qualify for this analysis. Of these 351 men, 39 had prior asbestos work employment elsewhere, 270 were dead, and 42 were lost to follow-up. Almost all of those host to follow-up were lost immediately after termination of their factory employment. Actually, this cohort exhibited decidedly unfavorable mortality even before the 20-year point (8, 9). Table 1 demonstrates that 582 men came under surveillance at the 20-year point and that the experience for each man from that time to December 31, 1977, included 6,311 man-years of observation. Table 1 also gives the age distribution of these men.

TABLE 1.—NUMBER OF MEN AT 20-YEAR POINT AND MAN-YEARS OF OBSERVATION, 1961–77, 20 OR MORE YEARS FROM ONSET OF EMPLOYMENT, 1941–45, FOR 582 AMOSITE ASBESTOS FACTORY WORKERS BY AGE

Age group years	Number of men	Man-years
35–39	84	150
40–44	99	663
45–49	90	1,062
50–54	86	1,192
55–59	65	1,037
60–64	55	787
65–59	40	560
70–74	35	406
75–79	20	266
80–84	6	126
85+	2	62
Total	582	6,311

Most of the men in this factory had a history of cigarette smoking. Table 2 outlines the smoking habits of the 582 men we have studied: 430 (73.9%) had a history of regular cigarette smoking (and almost 85% of these were still smoking at onset of observation), whereas 137 men had no such history. Of these 137 men, 59 (10.1%) had smoked pipes and/or cigars but not cigarettes, whereas only 78 (13.4%) had no history of any tobacco smoking. Smoking histories were unobtainable for 15 men (2.6%).

TABLE 2.—NUMBER OF MEN AT 20-YEAR POINT AND MAN-YEARS OF OBSERVATION, 1961–77, 20 OR MORE YEARS FROM ONSET OF EMPLOYMENT, 1941–45, FOR 582 AMOSITE ASBESTOS FACTORY WORKERS BY SMOKING HABITS AT 20-YEAR POINT

Smoking history	Number of men	Man-years
History of cigarette smoking	430	4,811
Current smokers	359	3,993
Exsmokers	71	818
No history of cigarette smoking	137	1,396
Never smoked regularly	78	868
Pipe and/or cigar only	59	528
Smoking history unknown	15	104

The entire cohort has been under surveillance since 1961. with the majority undergoing periodic clinical examination. For men for whom this periodic examination was not possible, intermittent contact was achieved. Whenever a death occurred, all available clinical and pathologic material was sought and reviewed, including reports of personal medical attendants, hospital records, and histologic material obtained at operation and/or autopsy. For each death a certificate of death was obtained.

Each individual in the cohort remained in the analysis "at risk" from the point at which he reached 20 years from onset of employment during the period 1961–65 until he took up other asbestos employment or otherwise until December 31, 1977, if he was still alive or until date of death if he died before then. The group as a whole then included 6,311 man-years of observation (table 1): 4,811 man-years for individuals with a history of cigarette smoking, 868 for those who never smoked regularly, 528 for those who had smoked pipes and/or cigars, and 104 for those whose smoking history was unknown (table 3).

TABLE 3.—NUMBER OF MEN AT 20-YEAR POINT AND MAN-YEARS OF OBSERVATION, 1961–77, 20 OR MORE YEARS FROM ONSET OF EMPLOYMENT, 1941–45, FOR 582 AMOSITE ASBESTOS FACTORY WORKERS BY AGE

Age group years	History of cigarette smoking		Never smoked regularly		Pipe and/or cigar only		Smoking history unknown	
	Number of men	Man-years	Number of men	Man-years	Number of men	Man-years	Number of men	Man-years
35–39	67	121	10	19	3	6	4	4
40–44	80	529	10	80	9	39	0	15
45–49	70	837	15	140	3	64	2	21
50–54	70	942	8	166	5	67	2	17
55–59	52	814	6	132	3	69	4	22
60–64	38	630	8	93	8	47	1	17
65–69	25	433	9	69	6	51	0	7
70–74	19	287	7	67	9	52	0	0
75–79	7	148	4	56	8	62	1	0
80–84	1	49	1	37	3	39	1	1
85+	1	21	0	9	1	32	0	0
Total	430	4,811	78	868	59	528	15	104

Using the attained age of each man in each calendar year period after admission to the cohort following the 20-year-from-onset point, 1961–77, we computed the number of expected deaths for this cohort for these periods of years.

Calculations were made in several ways. First, expected deaths were based on age-specific date of the U.S. National Center for Health Statistics for New Jersey white males (1963–66, 1967–71, and 1972–77) without regard to smoking habits. Expected deaths were also computed in relation to smoking habits on the basis of age- and smoking-specific experience (1963–66 and 1967–71) of white males in the ACS prospective study (10); this analysis was limited to the experience of men other than farmers with at most a high school education and a history of occupational exposure to dust, fumes, chemicals, gases, or radiation. The 1967–71 ACS study rates were extrapolated to 1972–77 according to changes in age-specific death rates among total U.S. white males between 1967–71 and 1972–77. Expected deaths for the few men with unknown smoking habits were computed on the basis of the age-specific and calendar-year-specific rates of all the men (regardless of smoking history) in the ACS study. A third set of expected deaths was computed on the basis of the death rates of the amosite asbestos factory workers who never smoked regularly. To ensure more stable figures, we initially used age-specific death rates for all of the men with experience past 20 years from onset of employment (regardless of smoking habits), 1961–66, 1967–71, and 1972–77. The resulting expected deaths were then proportionately adjusted to levels such that totals of expected and observed deaths were equal for the workers who never smoked regularly.

Observed deaths were then compared with deaths expected. We classified the causes of death in the observed deaths in two ways, according to DC and according to BE. Some investigators may prefer to use the DC figures and argue that the deaths in the populations used as controls have not been subject to a review similar to that of the BE deaths. Their preference assumes that whatever errors exist in the DC figures are much the same in the population being investigated (factory workers) as in the control populations (ACS or U.S. National Center for Health Statistics data). This procedure is certainly far less laborious.

However, some serious difficulties arise. When the distributions of causes of death in the population being investigated and control groups are markedly different, one might anticipate the distributions of error to be different. Mesothelioma and asbestosis have been decidedly underreported on the death certificates of asbestos workers. However, because of the infrequency of these conditions, they have not posed corresponding difficulties in the general population.

Whatever one does, one cannot ensure strict comparability between the cause-of-death classifications for the population investigated and the control populations. In the interest of showing as accurate results as we can for the population being studied, we have oriented the exposition in this paper to the BE findings. However, the DC results have also been shown in the tables for those who prefer such data.

Paralleling the observations for insulation workers (3, 11), some differences were observed among the various categories between cause of death as recorded on the death certificate and cause of death established after review (table 4). However, on

the whole, this set of findings speaks well for the diagnostic acumen of the media; attendants of these men.

RESULTS

Three hundred and four deaths occurred among the 582 men observed sometime between 1961 and 1965 (the point at which each man reached 20 yr from onset of employment) to December 31, 1977. Cancer of one or another site accounted for 116 or these deaths (38.2%), and 18 deaths were due to asbestos (5.9%). Table 5 shows that the following cancers in liver areas caused most of the deaths: lung cancer (60 deaths); pleural mesothelioma (7 deaths); peritoneal mesothelioma (7 deaths); gastrointestinal cancer including esophagus (16 deaths); and cancers of the oral cavity, pharyngeal, and laryngeal tissues (5 deaths). Altogether, there were approximately three and one-half times as many cancer deaths as expected. The increase in cancer deaths, coupled with deaths due to asbestos, serves to explain most of the virtual doubling of the expected deaths for these men over the period under study. In contrast to the insulation workers, the factory workers had an excess, though a limited one, in mortality from cardiovascular diseases (3, 11).

TABLE 4.—CATEGORIZATION OF 304 DEATHS, 1961–77, 20 OR MORE YEARS FROM ONSET OF EMPLOYMENT, 1941–45, FOR 582 AMOSITE ASBESTOS FACTORY WORKERS

Cause of death	Based on DC only	Based on BE	Difference between BE and DC
All causes	304	304	0
Cancer, all sites	103	116	+13
Lung	52	60	+8
Pleural mesothelioma	1	7	+6
Peritoneal mesothelioma	0	7	+7
Mesothelioma not specified above	3	0	−3
Larynx, oral cavity, and pharynx	3	5	+2
Esophagus	1	1	0
Kidney	2	2	0
Colon-rectum	11	11	0
Stomach	4	4	0
Prostate gland	4	4	0
Bladder	2	2	0
Pancreas	5	4	−1
Other and unspecified	15	9	−6
Noninfectious pulmonary dieseases, total	29	24	−5
Asbestosis	8	18	+10
Cardiovascular diseases	130	122	−8
Other and unspecified causes	42	42	0

Data in tables 6 and 7 are organized to shed some light on the excess deaths associated with amosite asbestos exposure, tobacco habits, and amosite asbestos plus tobacco exposures. Overall mortality in nonsmoking asbestos factory workers was more than doubled as compared with the experience of comparable men who had no asbestos exposure (table 6). This observation shows the strong influence of amosite asbestos exposure in itself.

For the total cohort of asbestos factory workers, if expected deaths had been calculated on the basis of the experience of asbestos factory workers who never smoked regularly, it could be estimated that 244 deaths would have occurred, whereas 91 deaths would have been anticipated for men who had neither a history of tobacco smoking nor asbestos employment (table 6). The influence of asbestos alone can be seen as the difference, or 153 deaths. Similarly, if expected deaths were based on men with the same smoking habits, but not with a history of asbestos employment, 137 deaths would have occurred instead of the 91 (table 6). The influence of smoking along would thus account for 46 deaths. The remainder of 14 when 91+153+46 is subtracted from the observed total of 304 deaths can be regarded either as the joint effect of asbestos and smoking or as discrepancies in data that do not purport to be absolutely consistent. The 304 deaths then are distributed as 30% neither asbestos related nor smoking related, 50% related to asbestos alone, 15% related to smoking alone, and 5% remaining.

Performing the same computation for the asbestos factory workers who were cigarette smokers, we estimated that of the 214 deaths (table 6), 57 (27%) were neither

asbestos nor smoking related, 115 (34%) were related to asbestos alone, 46 (21%) were related to smoking alone, and −4 (−2%) were the remainder.

Although these data can hardly be regarded as definitive, they do illustrate the general nature of the composite effects evident for all causes of death. The composite effects include, among others, specific smoking effects, asbestos effects—pleural mesothelioma, peritoneal mesothelioma, asbestosis, and the excess deaths from cancers of the stomach, colon-rectum, oral cavity, pharynx, and larynx as well as the limited number of excess deaths from lung cancer among the nonsmoking asbestos workers—and the very much larger increase in the lung cancer category as a result of the interaction between asbestos exposure and cigarette smoking.

TABLE 5.—OBSERVED AND EXPECTED DEATHS, 1961–77, 20 OR MORE YEARS FROM ONSET OF EMPLOYMENT, 1941–45, AMONG 582 AMOSITE ASBESTOS FACTORY WORKERS BY CAUSE OF DEATH

Cause of death	Observed deaths		Expected deaths	
	DC	BE	New Jersey [1]	Smoking adjusted: ACS study [2]
All causes	304	304	158.6	137.1
Cancer, all sites	103	116	33.4	29.8
Lung	52	60	10.1	10.1
Pleural mesothelioma	1	7	[3]	[3]
Peritoneal mesothelioma	0	7	[3]	[3]
Mesothelioma not specified above	3	0	[3]	[3]
Larynx, oral cavity, and pharynx	3	5	1.6	1.0
Esophagus	1	1	0.8	0.6
Kidney	2	2	0.8	0.9
Colon-rectum	11	11	5.2	3.7
Stomach	4	4	2.0	1.4
Prostate gland	4	4	2.7	2.5
Bladder	2	2	1.4	0.9
Pancreas	5	4	1.8	1.8
Other and unspecified	15	9	7.0	6.9
Noninfectious pulmonary dieseases, total	29	24	4.7	7.4
Asbestosis	8	18	[3]	[3]
Cardiovascular diseases	130	122	95.3	81.2
Other and unspecified causes	42	42	25.2	18.7

[1] Expected deaths were computed on the basis of age-specific data of the U.S. National Center for Health Statistics for New Jersey white males (1963–66, 1967–71, and 1972–77) without regard for smoking habits.

[2] Expected deaths were computed on the basis of age- and smoking-specific experience (1963–66 and 1967–71) of white males in the ACS prospective study. This analysis involved men with at most a high school education and with a history of occupational exposure to dust, fumes, chemicals, gases, or radiation; farmers were excluded from this group. The 1967–71 ACS study rates were extrapolated to 1972–77 according to changes in age-specific death rates in total U.S. white males between 1967–71 and 1972–77.

[3] New Jersey and ACS study death rates are not available, but these diseases have been rare causes of death in the general population.

TABLE. 6.—OBSERVED AND EXPECTED TOTAL DEATHS. 1961–77. 20 OR MORE YEARS FROM ONSET OF EMPLOYMENT. 1941–45. AMONG 582 AMOSITE ASBESTOS FACTORY WORKERS BY SMOKING HABITS AT 20-YEAR POINT

Smoking history	Observed deaths	Expected deaths based on		
		Amosite asbestos factory workers who never smoked regularly [1]	ACS study	
			Men with matching history of smoking [2]	Men who never smoked regularly [3]
History of cigarette smoking	214	171.9	102.3	56.7
Never smoked regularly	38	38.0	15.5	15.5
Pipe and/or cigar only	41	30.7	18.1	17.7
Smoking history unknown	11	3.0	1.2	0.8
Total	304	243.6	137.1	90.7

[1] Expected deaths were initially computed on the basis of age-specific experience (1961–66. 1967–71. and 1972–77) of all men in the cohort 20 or more yr from onset of exposure. The expected death totals were then adjusted proportionately so that the expected death total for the men who never smoked regularly equaled their observed death total.

[2] Expected deaths were computed on the basis of age- and smoking-specific experience (1963–66 and 1967–71) of white males in the ACS prospective study. The analysis was limited to men with at most a high school education and with a history of occupational exposure to dust, fumes, chemicals, gases, or radiation; farmers were excluded from this group. The rates in the ACS study (1967–71) were extrapolated to 1972–77 according to changes in age-specific death rates in total U.S. white males between 1967–71 and 1972–77.

[3] As in footnote 2, except that expected deaths were computed on the basis of the experience of the men who had never smoked regularly.

The last phenomenon, the remarkable multiplication of the already high lung cancer risk of cigarette smoking that occurs in asbestos workers, is clearly seen in table 7. The 0.2 deaths were expected to occur among the nonsmoking asbestos workers if their experience had been the same as comparable nonsmokers who had no history of asbestos work. However, 3 such deaths (BE classification) occurred among the 78 men in the group who had no history of regular cigarette smoking. This observation suggests that asbestos exposure alone produces an increased risk of lung cancer and agrees with observations in our cohorts of asbestos insulation workers (3). In terms of total number of lung cancer deaths, the increase is limited although undesirable. However, the increase in number of lung cancer deaths among cigarette smokers is devastating. Among 430 men with a history of cigarette smoking were 55 men who died from lung cancer (BE). approximately six times the number of other cigarette smokers without a history of asbestos employment and approximately 80 times the number expected among men of the same age and same personal background but without a history of either tobacco smoking or asbestos employment (0.7 expected and 55 observed).

TABLE 7.—OBSERVED AND EXPECTED LUNG CANCER DEATHS. 1961–77. 20 OR MORE YEARS FROM ONSET OF EMPLOYMENT. 1941–45, AMONG 582 AMOSITE ASBESTOS FACTORY WORKERS BY SMOKING HABITS AT 20-YEAR POINT

Smoking history	Categorization	Observed deaths	Expected deaths based on		
			Amosite asbestos factory workers who never smoked regularly [1]	ACS study	
				Men with matching history of smoking [2]	Men who never smoked regularly [3]
History of cigarette smoking	DC	45	28.8	9.6	.7
	BE	55	16.5	.....	.....
Never smoked regularly	DC	5	5.0	.2	.2
	BE	3	3.0	.....	.....
Pipe and/or cigar only	DC	2	3.0	.2	.2
	BE	2	1.8	.....	.....
Smoking history unknown	DC	0	.5	.1	0
	BE	0	.3	.....	.....
Total	DC	52	37.3	10.1	1.1
	BE	60	21.6	.....	.....

[1] Expected deaths were initially computed on the basis of age-specific DC and BE experience (1961–66. 1967–71. and 1972–77) of all men in the cohort 20 or more yr from onset of exposure. The expected death totals (DC BE) were then adjusted proportionately so that the expected death total for the men who never smoked regularly equaled their observed death (DC and BE).

[2] Expected deaths were computed on the basis of age- and smoking-specific experience (1963–66 and 1967–71) of white males in the ACS prospective study. This analysis was limited to men with at most a high school education and with a history of occupational exposure to dust, fumes, chemicals, gases, or radiation; farmers were excluded from this group. The rates in the ACS study (1967–71) were extrapolated to 1972–77 according to changes in age-specific death rates in total U.S. white males between 1967–71 and 1972–77.

[3] As in footnote 2, except that expected deaths were computed on the basis of the experience of the men who had never smoked regularly.

For other asbestos-related diseases, considerable interest exists in whether the risks of death associated with asbestos exposure are also influenced by cigarette smoking. However, there are only a small number of deaths at hand for use in investigation of these diseases among the amosite asbestos factory workers, especially among the nonsmoking workers.

Table 8 shows the data available for mesothelioma, gastrointestinal cancer, and asbestosis deaths among the factory workers (cigarette smokers and nonsmokers). To supplement these insubstantial findings, table 8 also gives corresponding data for the asbestos insulation workers (Hammond EC, Selikoff IJ, Seidman, H: Unpublished data.) The BE classification of causes of death is used because all comparisons in table 8 are within the asbestos worker groups. Moreover, the DC classification does not provide results of any value with respect to the incidence of deaths from mesothelioma and asbestosis.

We found no increased risk of death from mesothelioma in amosite asbestos factory workers with a history of cigarette smoking compared with nonsmokers. The same was true for cancer of the stomach, colon, and rectum. These observations agree with those for the asbestos insulation workers. However, the factory workers

do not show the strong influence of cigarette smoking on risk of death from asbestosis that was so striking in insulation workers (table 8).

TABLE 8.—OBSERVED AND EXPECTED DEATHS FROM SELECTED CAUSES (BE) 20 OR MORE YEARS FROM ONSET OF EMPLOYMENT FOR 582 AMOSITE ASBESTOS FACTORY WORKERS, 1961–77, AND 12,051 ASBESTOS INSULATION WORKERS, 1967–76, FOR MEN WITH A HISTORY OF SMOKING CIGARETTES COMPARED WITH THOSE WHO NEVER SMOKED REGULARLY

Causes of death	Cigarettes or NSR [1]	Amosite asbestos factory workers		Asbestos insulation workers	
		Observed deaths, BE	Expected deaths, NSR [2]	Observed deaths, BE	Expected deaths, NSR [3]
Mesothelioma	Cigarettes	9	14.8	105	108.8
	NSR	3	3.0	15	15.0
Cancer of stomach, colon-rectum	Cigarettes	9	10.1	43	41.5
	NSR	3	3.0	6	6.0
Asbestosis	Cigarettes	11	10.0	97	32.8
	NSR	2	2.0	5	5.0

[1] NSR=never smoked regularly.

[2] Expected deaths were initially computed on the basis of the age-specific BE experience (1961–66, 1967–71, and 1972–77) of all amosite asbestos factory workers 20 or more yr from onset of exposure. The expected death totals were then adjusted proportionately so that the expected death totals for the men who never smoked regularly equaled their observed death totals.

[3] Expected deaths were initially computed on the basis of the age-specific BE experience (1967–71 and 1972–76) of all asbestos insulation workers 20 or more yr from onset of exposure. The expected death totals were then adjusted proportionately so that the expected death totals for the men who never smoked regularly equaled their observed death totals.

CONCLUSIONS

The mortality experiences of amosite asbestos factory workers are much the same as those of asbestos-exposed insulation workers, i.e., remarkably increased risk of deaths from asbestosis and cancers of several sites (lung cancer; pleural mesothelioma; peritoneal mesothelioma; gastrointestinal cancer; and oral cavity, pharyngeal, and laryngeal cancers) (11, 12). Among men 20 or more years from onset of asbestos exposure, observed prospectively from a 20-year point reached 1961–65 to December 31, 1977, 304 deaths were seen, approximately twice the number expected. Eighteen deaths from asbestosis occurred. Excess deaths, however, were largely due to neoplasms. Neoplasms caused 116 deaths, three and one-half times the number expected. Sixty cancer deaths were from bronchogenic carcinoma and 14 more were due to either pleural or peritoneal mesothelioma.

Nevertheless, had it not been for cigarette smoking, many of the excess deaths would have been avoided. Asbestos alone would have accounted for an important increase, including some excess deaths from lung cancer, but not for most of the lung cancer deaths. Here asbestos exposure greatly multiplied the already high risk that would have been present with cigarette smoking alone. The combination of cigarette smoking and asbestos exposure among these amosite asbestos factory workers was calculated to have increased their risk of lung cancer death about 80 times as compared with like men of the same age who neither smoked cigarettes nor worked with asbestos. This increased risk is very much the same as that seen among asbestos insulation workers. This observation indicates that the increased risk of death from lung cancer among cigarette-smoking asbestos workers is a specific interaction rather than coincidental, and not, for example, the result of other agents in the environment of the construction trades.

The factory in which these men worked closed in 1954, and no further occupational exposure to asbestos occurred for most of the men. For most workers, asbestos employment ceased in 1945 at or before the end of World War II. In other studies (8, 9) we found that cessation of occupational exposure to asbestos in this factory was not accompanied by cessation of risk of asbestos-associated disease, including mesothelioma, gastrointestinal cancer, and asbestosis. This finding makes it all the more necessary to repeat an admonition previously made (1): Asbestos workers who smoke cigarettes should stop smoking immediately and those who do not smoke should never begin. Furthermore, all avoidable exposure to asbestos must be prevented.

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ASBESTOS EXPOSURE, CIGARETTE SMOKING AND DEATH RATES*

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Before 1966, there was evidence from a number of studies conducted in several different countries that people with a history of occupational exposure to asbestos dust are at risk of developing asbestosis and mesothelioma and at very high risk of lung cancer.[1-4] There was also evidence that mesothelioma or lung cancer resulting from asbestos exposure does not usually appear until many years after initial exposure.[5] Little more was known about the sometimes fatal effects of exposure to asbestos dust although there was some evidence that it increases the risk of cancer of the digestive tract.[3]

This investigation was started in 1966 primarily to obtain information on the combined effects of cigarette smoking and exposure to asbestos dust in respect to death from lung cancer and chronic noninfectious pulmonary diseases; to obtain further evidence concerning cancer other than mesothelioma and lung cancer; and to obtain information on the degree to which occupational exposure to asbestos dust increases total death rates from all causes combined. Some of the early findings have been published.[6,7]

In any study of the effects of exposure to asbestos dust it is necessary to have information on two groups of people: an exposed group and a nonexposed group usually called the "control group." Obviously, the two groups should be as alike as possible except in respect to asbestos exposure. In mortality studies, the total population is often used as the control group; and age-sex specific death rates as officially reported for the total population are compared with the age-sex specific death rates of asbestos workers. When this is done (and we have done it) it is only because of

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lack of availability of a more suitable control group. For this investigation, we could not use the total population as the only control group for the reason that we had to have information on the age-specific death rates of men with each of various types of smoking histories; and such information is not available for the total population. Therefore, we had to obtain a more suitable control group described later.

THE EXPOSED GROUP

This investigation was made possible by the splendid cooperation of the International Association of Heat and Frost Insulators and Asbestos Workers that has about 120 locals in the United States and Canada. Until recently, most of the material handled by these insulation workers contained asbestos. Therefore, we will refer to all of the members as asbestos workers. The union supplied us with their complete membership list and in 1966 we wrote to each member requesting him to complete a questionnaire containing a number of questions including those on his smoking habits and his use (or nonuse) of protective masks. Date of birth and date of entry into the trade were ascertained from union records.

All of these men have been traced through December 31, 1976 and copies of the death certificates of those who died have been obtained. Since cause of death recorded on a death certificate is not always accurate, we wrote to the doctor who signed the certificate requesting further details on diagnosis as well as to the hospitals in which treatment may have been given, requesting them to lend us histologic slides and/or x-ray films. Treating physicians, pathologists, hospitals were extremely cooperative in this matter.

Some of the men on the 1,966 membership list died before January 1, 1967 leaving 17,800 alive at that date (Table 1). Altogether 2,271 died in the ten-year period, January 1, 1967–December 31, 1976; the number of man-years of observation totaled 166,853; and the average age of the men during the ten-year period was 44.4. Of the 17,800 men, 5,117 had entered the trade 20+ years prior to January 1, 1967; and 1,573 of these died before January 1, 1977. Between January 1, 1967 and December 31, 1976, another 6,934 men passed the 20-year point since entering the trade; and 373 of these died in the interval between the time they passed the 20-year point and December 31, 1976. Thus a total of 5,117 + 6,934 = 12,051 men had either reached the 20-year point prior to January 1, 1967 or reached that point before January 1, 1977. The number of man-years of experience past the 20-year point totaled 77,391; the average age during observation was 53.8; and there were 1,946 deaths. This report is confined to the 77,391 man-years of observation and the 1,946 deaths just mentioned.

TABLE 1.—NUMBER OF MEN, MAN-YEARS OF OBSERVATION, OBSERVED DEATHS AND AVERAGE AGE DURING OBSERVATION, TOTAL, AND UP TO AND MORE THAN 20 YEARS FROM ONSET OF ASBESTOS EXPOSURE IN A COHORT OF ASBESTOS INSULATION WORKERS IN THE UNITED STATES AND CANADA, JANUARY 1, 1967–DECEMBER 31, 1976

	Total	Asbestos Exposures*	
		<20 years	20+ years
Number of men	17,800	12,683	12,051
Number of man-years	166,853	89,462	77,391
Number of deaths	2,271	325	1,946
Average age during observation	44.4	36.3	53.8

*As of January 1, 1967, 5117 men had reached the 20+ year point from onset of occupational exposure to asbestos dust. An additional 6934 men reached that point at some time between January 1, 1967 and December 31, 1976.

THE CONTROL GROUP

Starting on October, 1, 1959, 468,688 men and 610,206 women were enrolled in a long term prospective epidemiological study by volunteer workers of the American Cancer Society in 1121 counties in 25 states.[8] All of them were over 30 years old at that time and most of them were over 40. Upon enrollment, each subject answered a detailed questionnaire; and most of the survivors answered repeat questionnaires distributed in 1961, 1963, and 1965. During that time, death certificates were obtained on those who died; and when cancer was mentioned on a death certificate, the doctor who signed the certificate was requested to supply additional information on the cause of death and the basis of the diagnosis. Follow-up was then discontinued for six years.

Tracing of the subjects was resumed on October 1, 1971 and was continued through September 30, 1972. (We are still following subjects who were very old at that time). One of the major reasons we resumed the tracing was to obtain a suitable control group for the present investigation. Because of the extremely large number of deaths after 1965, it was not feasible for us to request doctors to supply us with additional information on cause of death.

The selection of the subjects was such that persons at the bottom of the social scale were almost entirely excluded (e.g., impoverished migrant workers, illiterates, illegal immigrants, prison inmates, etc). Persons in these categories are not members of the asbestos insulation workers union; but they are included in the mortality statistics of the United States, and their death rates are very high. On the other hand, unlike the asbestos workers, a large proportion of the male subjects was employed in sedentary occupations involving little or no physical exertion; and lack of exercise is associated with increased risk of coronary heart disease—the leading cause of death among makes in the United States. In contrast, men lacking a college education and with a history of occupational exposure to dust, fumes, gases, chemicals, or radiation are most likely to be in occupations involving physical exertion; and in this respect they are similar to members of the asbestos insulation workers union. Some such exposures increase the risk of cancer or pulmonary disease. Most of the members of the asbestos inoculation workers union were white and virtually none of them were farmers.

For these reasons we selected as a control group for this investigation all of the male subjects who met the following specifications; white, not a farmer, no more than a high school education, a history of occupational exposure to dust, fumes, vapors, gases, chemicals or radiation and alive as of January 1, 1967 and traced thereafter. There were 73,763 such subjects. They were classified according to their smoking histories and the age-specific death rates of each such class was computed.

There was a problem. Death rates in the control group were known for the period January 1, 1967 through September 30, 1972 while members of the asbestos insulation workers union were traced during the period January 1, 1967–December 31, 1976. According to official mortality statistics, death rates of the general population of the United States changed somewhat during the second five-year period January 1, 1972–December 31, 1976 compared with the first, January 1, 1967–December 31, 1971, increasing for some diseases and declining for other diseases. Under the assumption that these changes probably also applied to the control group, the death rates of the control group were extrapolated to take this into account. The principal effect of the extrapolation was to increase the death rates from lung cancer and decrease the death rates from heart disease during the last five years as compared with the first five years.

CAUSES OF DEATH

Because of the pressure to fill out a death certificate promptly so that the body may be moved, what is recorded on the certificate is not always the best available information on cause of death. For example, in the absence of the patient's physician, the certificate may be signed by a doctor who knows less about the case; or an autopsy may indicate that the tentative diagnosis of cause of death was incorrect; or a review of the evidence may indicate for example, that the patient died of peritoneal mesothelioma involving the pancreas rather than having died of primary carcinoma of the pancreas. Fortunately, we were able to obtain additional medical information on almost all of the asbestos workers who died; and in many instances histologic sections or x-ray films were loaned to us for further review.

The first column of figures in TABLE 2 shows the 1946 deaths classified underlying cause of death according to what was written on the death certificates, following as nearly as possible the coding rules used by the United States National Center for Health Statistics. The second column shows these same deaths classified according to the best available medical information, including, in many instances, review of histologic sections by pathologists with a special interest in mesothelioma and review of x-ray films by physicians with a special interest in asbestosis. In some instances, two or more pathologic conditions are present in such degrees that any one of them alone or the combined effects might have been responsible for death. Our procedure is to code up to three such conditions (i.e., diseases or injuries). Following international custom, we designate one of these as the underlying cause of death; but this is sometimes a highly arbitrary decision. The third column of figures in TABLE 2 shows the difference between the first column, DC (death certificate), and the second column BE (best evidence).

TABLE 2.—DEATHS OCCURRING 20+ YEARS AFTER ONSET OF OCCUPATIONAL EXPOSURE TO ASBESTOS DUST. CAUSE OF DEATH CODED IN TWO DIFFERENT WAYS

Underlying cause of death	Based on death certificates only (DC)	Based on best evidence available (BE)	Difference (BE)-(DC)
All causes	1,946	1,946	.....
Cancer, all sites	845	912	+67
Lung	397	450	+53
Pleural mesothelioma	23	61	+38
Peritoneal mesothelioma	24	109	+85
Mesothelioma not specified above	54	0	−54
Larynx, buccal and pharynx	21	27	+6
Esophagus	17	17	0
Kidney	15	16	+1
Colon-rectum	54	55	+1
Stomach	18	21	+3
Prostate	24	26	+2
Bladder	7	9	+2
Pancreas	46	21	−25
Other specified sites	110	83	−27
Primary site unknown	35	17	−18
Noninfectious pulmonary diseases, total	177	204	+27
Asbestosis	76	160	+84
Cardiovascular diseases	638	566	−72
Other and unspecified causes	286	264	−22
Subtotal—all causes except cardiovascular diseases	1,308	1,380	+72

There were major discrepancies in the comparative figures. According to death certificates 845 deaths were due to cancer, while according to the best evidence available 912 deaths were due to cancer, a difference of 67 deaths. On the other hand, according to DC, 638 deaths were due to cardiovascular diseases (including stroke) while according to BE only 566 deaths were due to cardiovascular diseases, a difference of 72 in the opposite direction.

Cancer of the pancreas accounted for 46 deaths (DC) or 21 deaths (BE). The best available medical information and review of histologic sections indicated that of the 46 deaths ascribed to primary cancer of the pancreas according to the death certificates, 15 were actually due to peritoneal mesothelioma, 6 were due to cancer of some other specified primary site and five to unknown primary site. One death attributed to myocardial infarction (DC) was probably due to primary cancer of the pancreas.

Cancer of other specified sites accounted for 110 deaths (DC) or 83 (BE) a difference of 27. Cancer in which the primary site was unknown was assigned 35 deaths (DC) or 17 deaths (BE). Additional medical evidence (sometimes from an autopsy) accounted for this difference.

Mesothelioma accounted for 101 deaths (DC) or 170 deaths (BE), a difference of 69. Most of the extra 69 deaths had been recorded on the death certificate as due to cancer of some other primary site or "cancer, primary site unknown."

The category labeled "noninfectious pulmonary diseases" includes chronic bronchitis, emphysema, pulmonary fibrosis, other nonspecific pulmonary diseases, and pneumoconiosis including asbestosis. This group of diseases caused 177 deaths (DC) or 204 deaths (BE) a difference of 27. The subcategory, asbestosis was termed the underlying cause of death for 76 (DC) or 160 (BE) of these deaths.

Observed vs. Expected Number of Deaths

Tables 3 and 4 show the 1946 observed deaths classified by cause of death in the two different ways previously discussed: from death certificates (DC) and according to the best evidence available (BE). Two different sets of expected numbers of deaths are shown: the expected number based on the age and smoking history specific death rates of men in the control group; and the expected number based upon the age-specific death rates of all white males in the United States as reported by the National Center for Health Statistics for the period 1967–1976. The age standardization was by five-year age groups in each of two five-year intervals of time (1967–1971 and 1972–1976). Thus altogether four sets of comparisons between observed and expected numbers of deaths are shown. This was done because there may be differences of opinion as to which set is preferable. In this analysis, in most

instances, we prefer comparisons of the expected deaths based on the control group rates with the observed deaths classified according to the death certificates. Our reasons are: the control group subjects were similar to the asbestos workers in respect to education and in respect to general type of work (i.e., work involving physical exercise and a history of exposure to dust etc.), smoking habits were taken into consideration, and although death certificates are not always accurate, when two groups are compared it is highly desirable that information on the causes of death come from the same source. However, the set of comparisons just mentioned leads to overestimation of the degree of association between exposure to asbestos and death from cancer of the pancreas while it underestimates the impact of mesothelioma and asbestosis. For these diseases, we prefer the comparisons where cause of death was classified according to the best evidence available (BE).

Two indices of association are shown: the mortality difference which is the observed number of deaths minus the expected number of deaths and the mortality ratio which is the observed number of deaths divided by the expected number of deaths.

The general findings are shown in TABLE 3. For total deaths (disregarding cause of death) the mortality ratios were 1.70 (control group) and 1.41 (U.S. white males) and the mortality differences were +798.0 and +570.0 respectively. For cancer of all sites combined, the mortality ratios ranged from 3.05 (U.S. white males, DC) to 3.52 (control group, BE) and the mortality differences ranged form +567.9 to +653.0. Thus, according to every comparison, total death rates and death rates from cancer were elevated in the asbestos workers.

The expected number of deaths from noninfectious pulmonary diseases was higher for the control group (68.2) than for U.S. white males (53.8). This is not surprising since men in the control group had a history of occupational exposure to dust, fumes, vapors, etc. The mortality ratios ranged from 2.60 (control group, DC) to 3.79 (U.S. white males, BE) and the mortality difference ranged from +108.8 to +150.2. Asbestosis clearly accounted for these mortality differences. However, it seems likely that many of these deaths were due to the combined effects of asbestosis and damage to the lung parenchyma resulting from cigarette smoking. Evidence on this will be shown later in this paper.

In contrast, the expected numbers of deaths from cardiovascular diseases were lower for the control group (660.1) than for U.S. white males (752.7). We do not know the reason for this, but it may have been due to the fact that men in the control group had a history of occupational exposures usually involving physical exercise (in contrast to clerical work). The mortality ratios ranged from .75 (U.S. white males, BE) to .97 (control group, DC) and the mortality differences ranged from −22.1 to −186.7.

Considering all causes of death except cardiovascular disease, the mortality ratios ranged from 2.10 (U.S. white males, DC) to 2.83 (control group, BE).

TABLE 4 shows the findings for cancer of several different sites. For lung cancer, the mortality ratios ranged from 4.24 (U.S. white males, DC) to 5.51 (control group, BE) and the mortality differences ranged from +303.3 to +368.3. Thus, no matter which of the various comparisons is considered to be the most reliable, it is clearly apparent that death rates from lung cancer were greatly increased in this group of asbestos workers—enough so as to cause a significant decrease in their life expectancy. From the standpoint of longevity, this was the most important effect of exposure to asbestos dust in a group of workers, the majority of whom had a history of cigarette smoking.

Death rates from mesothelioma are not available for either the control group or U.S. white males; but they are presumably extremely low in these two groups, so the expected numbers of deaths from mesothelioma were probably less than 0.1. Consequently the mortality ratios must be extremely high. The mortality difference ranged from a trifle less than 101 to a trifle less than 170. The former figure is certainly an underestimate while the latter figure, which is based upon histologic evidence as well as other evidence, is probably very close to correct. According to this evidence, 61 of the asbestos workers died of pleural mesothelioma and 109 died of peritoneal mesothelioma.

Cancer originating in the larynx, pharynx or buccal cavity is more readily diagnosed than cancer of some other sites; and in a previous study, deaths from cancer of these sites were generally reported correctly on death certificates. Therefore, we were surprised that according to the death certificates only 21 of the asbestos workers died of cancer of these sites, while according to the best available evidence 27 died of these cancers. The mortality ratios ranged from 1.60 (U.S. white males, DC)

to 3.60 (control group, BE). Two of the mortality differences are statistically significant at the level of $p < .05$; the other two are not.

TABLE 3.—OBSERVED AND EXPECTED NUMBER OF DEATHS OCCURRING 20+YEARS AFTER ONSET OF OCCUPATIONAL EXPOSURE TO ASBESTOS DUST

Underlying cause of death	Observed deaths		Control group †			U.S. white males		
	From ‡	Total	Expected deaths	Observed minus expected	Ratio observed/expected	Expected deaths	Observed minus expected	Ratio observed/expected
All causes	.....	1946	1148.0	+798.0	1.70	1376.0	+570.0	1.41
Cancer	DC	845	259.0	+586.0	3.26	277.1	+567.9	3.05
	BE	912	.....	+653.0	3.52	.....	+634.9	3.29
Cardiovascular	DC	638	660.1	−22.1	0.97	752.7	−114.7	0.85
	BE	566	.....	−94.1	0.86	.....	−186.7	0.75
Noninfections pulmonary (total)	DC	177	68.2	+108.8	1.60	53.8	+123.2	3.29
	BE	204	.....	+135.8	1.99	.....	+150.2	3.79
Asbestosis	DC	76	*	+76.0	.....	*	+76.0	.....
	BE	160	.....	+160.0	.....	.....	+160.0	.....
All other causes	DC	286	160.8	+125.2	1.78	292.3	−6.3	0.98
	BE	264	.....	+103.2	1.64	.....	−28.3	0.90
Subtotal all causes exc. cardiovascular	DC	1308	487.9	+820.1	2.68	623.3	+684.7	2.10
	BE	1380	.....	+892.1	2.83	.....	+756.7	2.21

* Control group and U.S. death rates not available but these have been rare causes of death in the general population.
† Like men in American Cancer Society prespective study (see text).
‡ DC means cause of death according to death certificate information. BE means cause of death according to best evidence available.

TABLE 4.—OBSERVED AND EXPECTED NUMBER OF CANCER DEATHS OCCURRING 20+ YEARS AFTER ONSET OF OCCUPATIONAL EXPOSURE TO ASBESTOS DUST

Underlying cause of death (cancers)	Observed deaths		Control group †			U.S. white males		
	From ‡	Total	Expected deaths	Observed minus expected	Ratio observed expected	Expected deaths	Observed minus expected	Ratio observed expected
Lung	DC	397	81.7	315.3	4.86	93.7	303.3	4.24
	BE	450	.....	368.3	5.51	.....	356.3	4.80
Mesothelioma	DC	101	*	101.0	.....	*	101.0	.....
	BE	170	.....	170.0	.....	.....	170.0	.....
Larynx, buccal and pharynx	DC	21	7.5	13.5	2.80	13.1	7.9	1.60
	BE	27	.....	19.5	3.60	.....	13.9	2.05
Esophagus	DC	17	5.1	11.9	3.34	6.5	10.5	2.64
	BE	17	.....	11.9	3.34	.....	10.5	2.64
Kidney	DC	15	8.5	6.5	1.76	7.0	8.0	2.15
	BE	16	.....	7.5	1.88	.....	9.0	2.30
Colon-rectum	DC	54	30.5	23.5	1.77	34.1	19.9	1.59
	BE	55	.....	24.5	1.81	.....	20.9	1.62
Stomach	DC	18	12.5	5.5	1.44	12.7	5.3	1.42
	BE	21	.....	8.5	1.68	.....	8.3	1.65
Postate	DC	24	18.3	5.7	1.31	19.7	4.3	1.21
	BE	26	.....	7.7	1.42	.....	6.3	1.32
Bladder	DC	7	6.7	0.3	1.04	8.6	−1.6	0.82
	BE	9	.....	2.3	1.34	.....	0.4	1.05
Pancreas	DC	46	16.0	30.0	2.87	15.5	30.5	2.96
	BE	21	.....	5.0	1.31	.....	5.5	1.35
Other and unspecified cancers	DC	145	72.1	72.9	2.01	66.3	78.7	2.19
	BE	100	.....	27.9	1.39	.....	33.7	1.51

* Control group and U.S. death rates not available but these have been rare causes of death in the general population.
† Like men in American Cancer Society prospective study (see text).
‡ DC means cause of death according to death certificate information. BE means cause of death according to best evidence available.

TABLE 5.—NUMBER OF STUDY GROUP MEN WHO ATTAINED AT LEAST 20 YEARS FROM ONSET OF OCCUPATIONAL EXPOSURE TO ASBESTOS DUST AT SOME TIME JANUARY 1, 1967–DECEMBER 31, 1976, BY AGE JANUARY 1, 1967, AND BY SMOKING HISTORY AS OF 1966

Ages as of Jan. 1, 1967	Total in age group	History of smoking (number of men)							
		Cigarettes*	Current cigarettes 20+/day	Current cigarettes <20/day	Ex-cigarettes 20+/day	Ex-cigarettes <20/day	Never smoked regularly	Pipe/cigar only	Unknown history
Total	12,051	6,841	3,708	764	1,823	378	891	488	3,831
25–29	339	174	105	29	31	9	27	6	132
30–34	1,662	844	531	100	160	43	142	66	610
35–39	2,610	1,423	863	161	313	65	198	84	905
40–44	2,151	1,336	818	114	329	58	143	69	603
45–49	1,526	923	487	94	265	49	107	61	435
50–54	1,250	792	398	74	253	44	84	46	328
55–59	960	558	248	70	193	33	65	40	297
60–64	693	388	132	63	134	35	50	44	211
65–69	413	203	75	27	74	16	33	31	146
70–74	255	108	28	15	42	15	29	29	89
75–79	111	60	17	12	18	6	5	5	41
80–84	52	19	4	4	5	4	3	5	25
85–	29	13	2	1	6	1	5	2	9

* Includes the cigarette smokers who did not specify the number smoked per day.

For cancer of the esophagus, the mortality ratios ranged from 2.64 to 3.34. The mortality differences ranged from +10.5 to +11.9 and are statistically significant. Without much doubt, exposure to asbestos dust increased the risk of this disease in a group of men, the great majority of whom smoked cigarettes, cigars or pipes.

Colon-rectum cancer was in third place (after cancer and mesothelioma) as a cause of cancer death among the asbestos workers. The mortality ratios ranged from 1.59 to 1.81. The mortality differences ranged from +19.9 to +24.5 and are statistically significant ($p<.05$).

For kidney cancer, the mortality ratios ranged from 1.76 (control group, DC) to 2.30 (U.S. white males, BE) and the mortality differences ranged from +6.5 to +9.0. Two of the mortality differences are statistically significant; the other two are not.

Death rates from stomach cancer have been declining rapidly in the United States and only 18 (DC) or 21 (BE) of the asbestos workers died of this disease. The mortality ratios ranged from 1.42 to 1.68. The mortality differences ranged from +5.3 to +8.5 and are not statistically significant ($p>.05$).

Mortality ratios for cancer of the prostate ranged from 1.21 (U.S. white males, DC) to 1.42 (control group, BE). The mortality differences are not statistically significant.

Very few of the asbestos workers died of bladder cancer and there is no indication that exposure to asbestos dust increases the risk of this disease.

The figures for cancer of the pancreas are interesting because so many of the deaths attributed to this disease on death certificates were actually due to mesothelioma. If the death certificate were to be believed, then it would appear that exposure to asbestos dust greatly increases the risk of cancer of the pancreas. Better evidence suggests that death rates from cancer of the pancreas may be slightly elevated in asbestos workers, but this is far from certain.

For cancer of all other sites combined (including cancer of unknown primary sites) the mortality ratios ranged from 1.39 (control group, BE) to 2.19 (U.S. white males, DC). The mortality differences ranged from +27.9 to +78.7 and are statistically significant.

SMOKING HABITS

TABLE 5 shows the age distribution of the asbestos workers as of January 1, 1967 and the smoking habits of those who answered the questionnaire in late 1966. It is confined to the 12,051 men who by January 1, 1967 had passed the 20-year point since entering the trade or who passed the point before January 1, 1977. Of the 8220 men who answered the questionnaire, 891 (11%) said that they had never smoked regularly; 488 (6%) had a history of pipe or cigar smoking but never smoked cigarettes regularly and; 6841 (83%) were either current or ex-cigarette smokers.

Some of the cigarette smokers also smoked pipes or cigars or had done so in the past.

Our first question related to the degree to which cigarette smoking increased the death rates of the asbestos workers. This is shown in TABLES 6A and 6B for all causes of death and for lung cancer identified as such from the best available medical evidence (BE) (the preferred classification here because all comparisons are within the asbestos workers group). The asbestos workers were first divided into five groups according to smoking habits: 1) smoked 20+ cigarettes a day; 2) smoked cigarettes regularly but <20 a day; 3) history of regular pipe or cigar smoking but never smoked cigarettes regularly; 4) never smoked regularly; and 5) smoking habits unknown. The 20-a day and the <20 a day cigarette smokers are each subdivided into four groups: "current" meaning that they were smoking cigarettes regularly in 1966; ex-cigarette smokers who had given up the habit for <5 years; ex-cigarette smokers who had given up the habit for 5 to 9 years; and ex-cigarette smokers who has given up the habit for 10+ years.

TABLE 6A shows the observed and expected number of deaths in each smoking category. The expected number for each particular category was calculated by applying the age-specific death rates of all the asbestos workers (regardless of smoking history) to the age-specific man-years of exposure to risk for that particular smoking category. TABLE 6B, which is based upon TABLE 6A, shows two different ratios: the mortality ratio calculated by dividing the observed number of deaths by the expected number of deaths (by definition, the mortality ratio for all of the subjects is 1.00), and the ratio relative to never smoked regularly, NSR, which is the mortality ratio for each smoking category divided by the mortality ratio for men who never smoked regularly (by definition this ratio for men who never smoked regularly is 1.00). The latter ratio is essentially the ratio which would have been obtained if men who had never smoked regularly had been used as a control group; but this method of calculation provides somewhat more stable figures.

TABLE 6A

RVED AND EXPECTED NUMBER OF DEATHS OCCURRING 20+ YEARS AFTER ONSET OF UPATIONAL EXPOSURE TO ASBESTOS DUST BY SMOKING HISTORY. EXPECTED DEATHS RE BASED ON AGE-SPECIFIC DEATH RATES OF ALL OF THE ASBESTOS WORKERS

Smoking History	All Causes of Death		Lung Cancer (BE)†	
	Observed Deaths	Expected Deaths	Observed Deaths	Expected Deaths
tal*	1946	1946.0	450	450.0
+ cigarettes/day				
Current	565	481.3	171	116.8
Ex, <5 yrs.	166	137.5	54	33.3
Ex, 5–9 yrs.	61	69.6	10	17.1
Ex, 10+ yrs.	112	136.5	15	31.4
20 cigarettes/day				
Current	143	127.6	38	29.5
Ex, <5 yrs.	22	20.0	5	4.8
Ex, 5–9 yrs.	10	11.5	1	2.5
Ex, 10+ yrs.	31	43.6	5	9.8
pe/cigar only	86	112.5	3	24.2
ver smoked regularly	94	158.2	5	35.5
known	614	587.6	136	132.6

TABLE 6B

RTALITY RATIOS OF OBSERVED AND EXPECTED NUMBER OF DEATHS PRESENTED IN BLE 6A RELATIVE TO TOTAL MEN AND TO THE MORTALITY RATIOS FOR MEN WHO NEVER SMOKED REGULARLY (NSR)

Smoking History	All Causes of Death		Lung Cancer (BE)†	
	Ratio Observed/ Expected	Ratio to NSR	Ratio Observed/ Expected	Ratio to NSR
tal*	1.00	1.68	1.00	7.10
+ cigarettes/day				
Current	1.17	1.98	1.46	10.40
Ex, <5 yrs.	1.21	2.03	1.62	11.51
Ex, 5–9 yrs.	0.88	1.48	0.58	4.15
Ex, 10+ yrs.	0.82	1.38	0.48	3.39
20 cigarettes/day				
Current	1.12	1.89	1.29	9.15
Ex, <5 yrs.	1.10	1.85	1.04	7.40
Ex, 5–9 yrs.	0.87	1.46	0.40	2.84
Ex, 10+ yrs.	0.71	1.20	0.51	3.62
pe/cigar only	0.76	1.29	0.12	0.88
ver smoked regularly	0.59	1.00	0.14	1.00
known	1.04	1.76	1.03	7.28

addition to the smoking groups shown, the total includes the cigarette smokers who did not y the number smoked per day, or the length of time stopped if an ex-smoker.
ed of lung cancer according to the best evidence available.

The pattern of relationships between smoking habits and death rates was found to be essentially the same for asbestos workers as for other groups of men as previously reported by numerous different investigations.

The following applies to deaths from all causes combined; and the mortality ratios relative to NSR (never smoked regularly) (see TABLE 6B). The mortality ratios were 1.98 for men who currently smoked 20+ cigarettes a day, 1.89 for those who currently smoked <20 cigarettes a day, 1.29 for pipe and cigar smoking, and 1.00 for men who never smoked regularly. The mortality ratio for ex-cigarette smokers who previously smoked 20+ cigarettes a day and had given up the habit <5 years was 2.03. This is higher than the mortality ratio for men who currently smoked 20+ cigarettes a day. It is a usual finding and is almost certainly due to the fact that men with incurable lung cancer or other advanced disease often give up smoking a few months or longer before they die. The 20+ a day cigarette smokers who had given up the habit for 5 to 9 years had a mortality ratio of 1.48 and those who had quit for 10+ years had a mortality ratio of 1.38, so their death rates were lower than the death rates of men who currently smoked 20+ cigarettes a day. The mortality ratios for ex-cigarette smokers who had previously smoked <20 cigarettes a day were: 1.85 (quit <5 years), 1.46 (quit 5 to 9 years), and 1.20 (quit 10+ years) as contrasted with a mortality ratio of 1.89 for men who currently smoked <20 cigarettes a day.

For lung cancer, the pattern of relationship was about the same as described above, but the mortality ratios of cigarette smokers were far higher. For example, the mortality ratio was 10.40 for men who currently smoked 20+ cigarettes a day and 9.15 for men who currently smoked <20 cigarettes a day.

There were too few deaths from each of various other diseases to present such a detailed analysis in relation to smoking habits. Some of the findings are briefly described below using the ratio Observed/Expected.

Only five men who never smoked regularly died of noninfectious pulmonary diseases whereas 17.1 deaths were expected in this group (ratio .29). These five died of asbestosis. Thus, for the subcategory asbestos, there were five deaths among men who never smoked regularly whereas 13.3 were expected (ratio .38). Since all of the other men were smokers (mainly cigarette smokers) it follows that death rates from asbestosis were far higher in cigarette smokers than in nonsmokers.

The asbestosis death rates of men who currently smoked 20+ cigarettes a day was 2.8 times as high as the asbestosis death rate of men who never smoked regularly. It is clear that cigarette smoking greatly increases the risk of an asbestos worker dying from asbestosis or asbestosis combined with pulmonary fibrosis and emphysema resulting from cigarette smoking.

None of the men who had never smoked regularly died of cancer of the esophagus, larynx, pharynx or buccal cavity. The expected number of deaths from these cancers was 3.6 for men who never smoked regularly. This suggests that in the absence of exposure to tobacco, exposure to asbestos dust may have little or no influence on death rates from such cancers.

For pleural mesothelioma, there were three observed deaths vs. 4.8 expected deaths among men who never smoked regularly (ratio .63); and five observed deaths vs. 3.2 expected deaths for pipe/cigar smokers (ratio 1.59). Thus, for men who never smoked cigarettes regularly there were eight observed deaths and eight expected deaths from this disease; so it appears that cigarette smoking has little or no effect upon death rates from pleural mesothelioma.

OTHER COMPARISONS

In TABLE 7A, the observed number of lung cancer deaths among asbestos workers in each of several different smoking categories is contrasted with the number expected if their age-specific lung cancer death rates had been the same as those for control group subjects who never smoked regularly. The observed numbers are based upon death certificate information. The table is confined to asbestos workers whose smoking habits were known.

Altogether, according to the death certificates, 276 lung cancer deaths occurred among the asbestos workers whose smoking habits were known, whereas only 6.0 were expected had their age-specific lung cancer death rates been the same as those for control group subjects who never smoked regularly. Thus, the mortality difference was 270.0 and the mortality ratio was 46.23.

The mortality ratios for various smoking categories were 87.36 for asbestos workers who currently smoked 20+ cigarettes a day; 50.82 for those who currently smoked <20 cigarettes a day; 36.56 for ex-cigarette smokers; 7.02 for pipe/cigar smokers; and 5.33 for asbestos workers who never smoked regularly.

Taken at face value it would appear that the risk of dying of lung cancer is about five times as great for a nonsmoker occupationally exposed to asbestos dust as for a nonsmoker without such exposure. However, because of small numbers the ratio is subject to considerable statistical sampling variation. By chance, the observed number might have been as low as one or two or as high as about nine. Therefore, all we can say is that the actual mortality ratio is probably >1.00 but could be as high as 12.00 or even a little higher. In any event, the mortality ratio for men who never smoked regularly was very small as compared with the mortality ratios of the cigarette smokers.

The mortality differences shown in TABLE 7B are copied directly from TABLE 7A. Altogether, 270.0 more of the asbestos workers died of lung cancer than would have died had their lung cancer death rates been the same as for nonsmokers not exposed to asbestos dust. These may be called "extra" lung cancer deaths. Of these "extra" deaths, 55.7% occurred in asbestos workers who currently smoked 20+ cigarettes a day; 11.3% in those who currently smoked <20 cigarettes a day; 29.9% in ex-cigarette smokers; 1.3 percent in pipe/cigar smokers; and 1.21 percent in asbestos workers who never smoked regularly. These figures are shown to give some idea of the degree to which the lung cancer death rate of a group of asbestos workers depends upon their distribution in regard to smoking habits.

TABLE 7A.—OBSERVED NUMBER OF LUNG CANCER DEATHS OCCURRING 20+ YEARS AFTER ONSET OF OCCUPATIONAL EXPOSURE TO ASBESTOS DUST BY SMOKING HISTORY COMPARED WITH EXPECTED NUMBER BASED ON LUNG CANCER DEATH RATES OF CONTROL GROUP SUBJECTS WHO NEVER SMOKED REGULARLY

Smoking history	Lung cancer (DC)*			
	Observed	Expected	Mortality differences	Ratio observed/expected
Total (with smoking habits known)	276	6.0	+270.0	46.23
History of cigarettes †	268	4.7	+263.3	57.63
Current 20+ cigarettes/day	152	1.7	+150.3	87.36
Current <20 cigarettes/day	31	0.6	+30.4	50.82
Ex-cigarettes	83	2.3	+80.7	36.56
Pipe/cigar only	4	0.6	+3.4	7.02
Never smoked regularly	4	0.7	+3.3	5.33

TABLE 7B.—DISTRIBUTION OF LUNG CANCER MORTALITY DIFFERENCES PRESENTED IN TABLE 7A BY SMOKING HISTORY

Smoking history	Lung cancer (DC)*	
	Mortality differences	Percent of total
Total (with smoking habits known)	+270.0	100.0
History of cigarettes †	+263.3	97.5
Current 20+ cigarettes/day	+150.3	55.7
Current <20 cigarettes/day	+30.4	11.3
Ex-cigarettes	+80.7	29.9
Pipe/cigar only	+3.4	1.3
Never smoked regularly	+3.3	1.2

*Died of lung cancer according to death certificate information.
†Includes the current cigarette smoking asbestos workers who did not specify the number smoked per day.

TABLE 8 differs from all of the other tables in that it shows lung cancer death rates per 100,000 man-years standardized for age in the age distribution of all of the asbestos workers in this study. The lung cancer deaths were so reported on death certificates. Just four groups are shown identified by occupational exposure to asbestos dust, (yes or no) and history of cigarette smoking (yes or no). The word "no" under the "history of cigarette smoking?" means never smoked regularly (pipe and/or cigar only smokers being excluded from this table).

The mortality differences shown here were calculated by subtracting the death rate of the "no, no" group from the death rate of each of the four groups. The mortality ratios were calculated by diving the death rate of each group by the death rate of the "no, no" group.

The mortality ratios are 1.00 for "no, no," (asbestos no; cigarette smoking, no); 5.17 for "yes, no" (asbestos, yes; cigarette smoking, no); 10.85 for "no, yes" (asbestos, no cigarette smoking, yes); and 53.24 for "yes, yes" (asbestos, yes; cigarette smoking, yes).

Now, suppose that occupational exposure to asbestos dust and cigarette smoking acted independently in respect to the production of lung cancer. In that event, the lung cancer death rate of asbestos workers with a history of cigarette smoking should be very close to the sum of the following three numbers: 11.3 (the rate for the "no, no" group), 47.1 (the mortality difference for the "yes, no", group), and 111.3 (the mortality difference for the "no, yes" group). The sum comes to 169.7 lung cancer deaths per 100,000 man-years which is a reasonable estimate of what the lung cancer death rate of the asbestos workers with a history of cigarette smoking would have been if there had been no synergistic effect of the combined exposure. In contrast, the observed lung cancer death rate of the "yes, yes" group was 601.6 per 100,000 man-years. The difference, (601.6–169.7)—431.9 lung cancer deaths per 100,000 man-years, was presumably due to a synergistic effect in men with both of the two types of exposure (asbestos dust and cigarette smoking).

These particular figures apply to the particular group of asbestos workers included in this study. The net synergistic effect would not have been the same if their smoking habits had been different, and it probably would not have been the same if their lapsed times from first exposure to asbestos dust had been different or if the amount of asbestos dust they inhaled had been different.

TABLE 8.—AGE-STANDARDIZED LUNG CANCER DEATH RATES* FOR CIGARETTE SMOKING AND/OR OCCUPATIONAL EXPOSURE TO ASBESTOS DUST COMPARED WITH NO SMOKING AND NO OCCUPATIONAL EXPOSURE TO ASBESTOS DUST

Group	Exposure to asbestos?	History cigarette smoking	Death rate	Mortality difference	Mortality ratio
Control	No	No	11.3	0.0	1.00
Asbestos workers	Yes	No	58.4	+47.1	5.17
Control	No	Yes	122.6	+111.3	10.85
Asbestos workers	Yes	Yes	601.6	+590.3	53.24

*Rate per 100,000 man-years standardized for age on the distribution of the man-years of all the asbestos workers. Number of lung cancer deaths based on death certificate information.

COMMENTS

Underlying cause of death cannot always be ascertained with certainty; when cancer is clearly the cause of death, the primary site of the disease cannot always be ascertained with accuracy; and even when histologic sections are available, different pathologists may disagree on the histologic type of a cancer. These difficulties are troublesome from the standpoint of scientific accuracy as to details, but they make little difference to the workers and their families. For example, both peritoneal mesothelioma and cancer of the pancreas are painful diseases and the degree of pain is not dependent upon ascertaining which of these caused the death of the patient. If inadequate information results in error as to which of these two cancers caused death, then it only means that the degree of association between death rates and exposure to asbestos dust is overestimated for one of the two diseases and correspondingly underestimated for the other.

It is true that some diseases are more painful than others and some are more expensive than others. But what is of most importance to the worker and most important to society is whether a particular type of occupational exposure leads to premature death or often leads to long term disability. Occupational exposure to asbestos dust results in both of these. This is sufficient reason to take strong preventive measures regardless of further details which are of scientific interest or which can have an influence on the outcome of a law suit.

Evidence from this study indicates a strong synergistic effect between two types of exposure (asbestos and cigarette smoking) in respect to risk of lung cancer.

Evidence from this study indicates that among asbestos workers, ex-cigarette smokers have substantially lower death rates than cigarette smokers who do not give up the habit. This should be brought forcefully to the attention of present asbestos workers. A young person who is so strongly addicted to cigarette smoking that he cannot break the habit or is unwilling to do so would be particularly well advised not to enter a trade involving exposure to asbestos dust.

Cigarette smoking increases the risk of death from asbestosis, providing another reason why asbestos workers should avoid that habit.

The major specific fatal effects of occupational exposure to asbestos dust are increased death rates from lung cancer, mesothelioma and asbestosis. The evidence indicates that occupational exposure to asbestos dust increases death rates from cancer of the colon-rectum, and cancer of the esophagus. It probably increases death rates from cancer of the kidney and from another set of cancers taken as a group (i.e., larynx, pharynx and buccal cavity). Aside from this, the evidence suggests that death rates from cancer of several other sites may be somewhat increased by occupational exposure to asbestos dust. Because of small numbers, we cannot identify which, if any, other specific sites are involved. As will be published in the near future by another group of investigators, asbestos bodies have been found in many different organs of asbestos workers who died and whose lungs were found to contain a very large number of asbestos bodies.[10]

It must be emphasized that only one group of asbestos workers is included in this investigation, namely members of the insulation workers union. We have indicated the degree of confidence we have in various specific findings, and in many instances we have given more than one estimate. For example, we said that "the mortality ratios for colon-rectal cancer ranged from 1.59 to 1.81," the mortality ratios having been calculated from each of four different sets of figures. In this particular case, all four sets of comparisons led us to the conclusion that death rates from colon-rectal cancer are increased by exposure to asbestos dust; but it would be folly to suppose that we have precisely determined the degree of association even in this group of asbestos workers.

Aside from this, the degree of association between exposure to asbestos dust and death rates from all causes combined as well as death rates from lung cancer vary with smoking history, lapsed time from onset of exposure to asbestos dust," presumably with amount of asbestos dust inhaled, and probably with other factors as well. It seems very unlikely that any two identifiable groups of people with occupational exposure to asbestos dust are exactly alike in respect to all of the factors just mentioned. Therefore, we would not expect the exact figures reported here to be duplicated in a study of any other group of asbestos workers. We would expect the findings to be qualitatively the same but quantitatively identical.

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Mr. TRAFICANT. Anything else, Mr. Clyburn?

Mr. CLYBURN. No, sir.

Mr. TRAFICANT. I have a few more questions and then we will get on with the last witness.

Let me see if I can get just a few things established before we move on. The general perception in the legal community and people outside of Congress is that when people line up to appeal for a workers compensation case, they usually get it, it's damned near automatic, and when they do get it, they're on it forever. That is the assumption that exists. I'd like to cut through the assumption. How many of your workers compensation cases that are contested are in fact granted?

Mr. ROGERS. About half.

Mr. TRAFICANT. So the reports we have that it could be as high as 90 percent, is untrue?

Mr. ROGERS. Could I just tell you a little bit about the figures on this? On average, about probably 70 or 80 percent of the people who have a workers comp claim are out of work about 8 days, on average. The vast majority of the people have relatively minor injuries, they're off for a few days and go back to work. Very few cases turn into long-term disability, less than 10 percent.

Mr. TRAFICANT. On the ones that do, what is the percentage of the workers compensation awards?

Mr. ROGERS. The Federal Government system pays 66⅔ percent of the salary, your gross salary at the time of your injury without a dependent; 75 percent if you have one or more dependents, tax-free.

Mr. TRAFICANT. We talked earlier about the number of cases that are presented, we talked about the cost and the burden of proof and the cost that's presented on behalf of the worker filing such action. Is there not, in fact, a cost that is borne by the Government to answer these contested charges and to, in fact, deal with this contested arena of workers compensation rights?

Mr. ROGERS. I think it's the responsibility of the Government to provide a safe workplace, without a doubt. I think the Congress has charged the Executive Branch with that and I think the Executive Branch has moved in that direction over the past 10 years.

Mr. TRAFICANT. But I haven't asked you that. The worker that files a workers compensation case must prove it happened in the workplace. The heart attack doesn't necessarily mean because they fell over a desk at Agriculture, that it was the corn and soybeans in the accounting department that caused them to fall over. So they have that cost and they have to prove that they were debilitated and deserve compensation because it happened in the workplace. So they have that cost to bear, correct?

Mr. ROGERS. That's correct.

Mr. TRAFICANT. We then, by the very nature of that problem, being contested or being brought to us, have an accompanying cost. It costs us money to resolve that problem. Is that a correct statement?

Mr. ROGERS. That is correct.

Mr. TRAFICANT. Even if there is not, in fact, a compensation award?

Mr. ROGERS. Yes, sir.

Mr. TRAFICANT. What is the average cost to the Federal Government in dealing with a contested award?

Mr. ROGERS. I would have to try and make a calculation of that. I've never been asked that question before and it would be very difficult.

Mr. TRAFICANT. We're asking you now, Mr. Rogers.

Mr. ROGERS. And I'll be glad to try and answer it for you. We would do staff costs and things like that. We can answer the question, I've just never been asked before.

Mr. TRAFICANT. We will leave the record open and I would ask unanimous consent that specific economic question be submitted to this subcommittee so it can be distributed to everybody, including Mr. Valentine. Without objection, so ordered.

[The following was received from Mr. Rogers:]

FECA COSTS FOR CONTESTED CASES

We have been asked to determine the cost to the Office of Workers' Compensation Programs (OWCP) in dealing with a contested case. Since this cost varies widely between cases, and because we do not capture data about contested cases directly, we have chosen to do this in two steps. First, we estimated the percentage of staff time devoted to contested cases and applied this percentage to our annual administrative budget to arrive at an estimate of the annual total cost for all contested cases. Second, we estimated the total number of contested cases and divided this figure into the annual cost figure to arrive at the cost per case.

Since the post-adjudication course of contested and non-contested cases is identical, we focused on the cost of adjudicating contested cases, basing our estimate on a time study performed in 1991. This study categorized Division of Federal Employees' Compensation (DFEC) employees' activities and measured the amount of time spent at each activity, expressing the result as a percentage of total staff time. From the categories used in this study, we identified those activities which support the adjudication of all cases and made an estimate of the fraction of the activity which can be attributed to adjudicating contested cases. Multiplying the percentage by the fraction gives a percentage of staff time devoted to that activity in connection with the adjudication of contested cases. Adding up the percentages gives the total percentage of staff time devoted to the adjudication of contested cases. (See attachment.)

The resulting percentage is 20.2%. Multiplying this percentage by the DFEC Fiscal Year 1992 budget of $60,337,000 gives a figure of approximately $12,188,000 as the cost to OWCP of adjudicating contested cases.

To estimate the number of contested cases, we considered traumatic injury cases and occupational disease cases separately. For traumatic injuries, we calculated the percentage of "Continuation of Pay" cases that were controverted by the employing agency during 1992 and multiplied this percentage by the total number of traumatic injury cases received, giving an estimate of 11,884 traumatic injury cases contested. For occupational diseases, we assumed that 75% of all occupational disease cases are contested, or 16,827 of the occupational disease cases received during 1992. Thus, we estimate that 28,708 contested cases were received during 1992.

Dividing the $12,188,000 estimate of the annual cost of adjudicating contested cases by the estimated 28,708 contested cases received per year results in an estimated cost to OWCP of $424.55 to adjudicate a contested case.

ESTIMATED PERCENTAGE OF STAFF TIME SPENT ADJUDICATING CONTESTED CASES—

Activities associated with contested claims	Activity as a percentage of all work	Activity fraction for contested claims	Contested claims activity as a percentage of all work
Traumatic adjudication	9.6	.50	4.8
Occ. dis. adjudication	4.9	.75	3.7
Remands	0.5	.40	0.2
Medical referrals	4.1	.13	0.5
Compensation orders	2.2	.50	1.1
Telephone inquiries	12.0	.25	3.0
Priority inquiries	4.6	.33	1.5

ESTIMATED PERCENTAGE OF STAFF TIME SPENT ADJUDICATING CONTESTED CASES—
Continued

Activities associated with contested claims	Activity as a percentage of all work	Activity fraction for contested claims	Contested claims activity as a percentage of all work
Miscellaneous	26.5	(1)	5.3
Total	[2] 64.4		20.2

[1] Miscellaneous activities are prorated to contested claims in the same ratio as the specified activities bear to non-miscellaneous activities.
[2] Remainder of work activity is related to post-adjudication issues such as bill and compensation payment and medical management.

Mr. TRAFICANT. Mr. Valentine?

Mr. VALENTINE. Mr. Chairman, I hesitate to insinuate myself in this but, in line with what you've said, I would like to ask a question, if I might.

Aren't a large number of these workers comp cases handled without benefit of an attorney?

Mr. ROGERS. In the Federal system, that is correct.

Mr. VALENTINE. About how many, percentagewise?

Mr. ROGERS. Looking at the system, we think less than 5 percent of the claimants have representation of any kind.

Mr. VALENTINE. Less than 5 percent?

Mr. ROGERS. Yes, sir.

Mr. VALENTINE. A lot of them are handled in a more or less routine fashion, aren't they?

Mr. ROGERS. Yes, sir.

Mr. VALENTINE. When you gave the distinguished Chairman the statistics about workers comp, you were just talking about the Federal workers comp in the 50 States?

Mr. ROGERS. Not the 50 States, only the Federal.

Mr. VALENTINE. There might be a big difference in the workload and the way the State apparatus or machinery addresses this problem?

Mr. ROGERS. Indeed, that's the case. Many States do litigate. This is an administrative process in the Federal Government.

Mr. VALENTINE. Thank you, Mr. Chairman.

Mr. TRAFICANT. In most of these cases, someone could fall and get hurt in the Rayburn Building and file a workers comp case and even a liability case if there was some water left over and there could be attorneys, and we can deal with asbestos, we can deal with maybe radon at this point, but basically now, there is a new litigation instrument available for workers comp cases.

Would you agree that ETS now, by the nature of the findings, puts it in a position where there could be an increase to the Federal Government in these types of claims being presented?

Mr. ROGERS. It's possible, but the exposure is going down, as I've got to say. I have to balance the equation. Clearly, factually, the exposure is going down.

Mr. TRAFICANT. Good.

Any further questions?

[No response.]

Mr. TRAFICANT. I thank the witnesses and we will be sending you some questions, I'm sure.

Our last witness is associated with the cause of much of this controversy. People in some quarters are tarring and feathering this agency. I have, on occasions, recommended that we straighten out our balance of trade by sending the EPA to Japan. [Laughter.]

We are here to talk about someone who can answer some questions about the EPA study, Dr. Douglas W. Dockery, Associate Professor, Department of Environmental Epidemiology, Harvard School of Public Health, Boston, Massachusetts. Doctor, we welcome you. We ask that you summarize so we can question you. Go right ahead.

TESTIMONY OF DR. DOUGLAS W. DOCKERY, ASSOCIATE PROFESSOR, DEPARTMENT OF ENVIRONMENTAL EPIDEMIOLOGY, HARVARD SCHOOL OF PUBLIC HEALTH

Dr. DOCKERY. I appreciate the invitation to come here and speak. I appreciate the committee being present. I'm truly flattered that Mr. Valentine has come specifically to hear my talk. I consider myself a poor country practicing epidemiologist and I hope I can respond to his concerns and also the concerns of the rest of the committee.

Indeed, I do have the title of Associate Professor of Environmental Epidemiology at the Harvard School of Public Health. My research over the past 20 years there has been focused on respiratory health associated with indoor air pollution and outdoor pollution. I've had a continuing interest in environmental tobacco smoke and its exposures and the effects it has on children. It really has been one of the major focuses of my research over the years, and also an interest in the effects of smoking, both active and passive, on the development of chronic obstructive lung disease and other nonmalignant respiratory diseases in adults.

I teach environmental epidemiology at the School of Public Health to graduate students there. I teach environmental and occupational epidemiology during this term. I just had my class yesterday. I teach respiratory epidemiology in the fall. In both cases, we use these materials that we're talking about as examples in the class.

I was asked to testify before the EPA hearings by the Science Advisory Board two years ago and again last year by the Coalition on Smoking or Health and prepared testimony at that time. I've had a chance to review this EPA document in a lot of detail. It's been well-used and this is my second copy that I've gone through and you can see even this one is beginning to get a little dog-eared.

I've prepared written critiques and I've sent those in and in fact, last week in my class, we used this document as the basis of the class that we conducted and we have gone through this in a lot of detail.

I've submitted my written testimony. I'd like to couch my remarks in terms of some of the things that have been said previously this morning.

I personally find this to be a real extraordinary document, the EPA Review of the Health Effects of Passive Smoking. This is a comprehensive and balanced and very scholarly document, that's been prepared by the EPA. As I said, I had a chance to submit my

critiques of this document in the preparation stage. I think it certainly better in response to those, but it is a very good document.

As opposed to Dr. Batta Gori, who characterized this as being a contradictory and uninterpretable document and reviewing contradictory and uninterpretable studies, I think this really has been a shining example of how the review of epidemiologic data should be done.

I was very interested when you asked the question regarding positive effects of tobacco smoke and the studies were mentioned regarding positive effects associated with uterine cancer, colon cancer, prostate cancer, Parkinson's disease, colitis. I have no doubts that there are quality studies that have been done suggesting that there are some benefits associated with tobacco smoke associated with these specific cancers. They are probably very good studies and very important to us in understanding what the mechanisms are associated with those specific diseases.

On the other hand, I think that the balance of evidence is clearly that tobacco smoke is a very strong carcinogen in the net. Being one who is beginning to look to the future and realize that I am at risk for prostate cancer and colon cancer and Parkinson's disease in the future, I don't think I would take the intervention of starting to smoke now to reduce my risk for those particular diseases.

It's interesting that these studies are cited because they are, I'm sure, epidemiologic evidence of the same quality of that which is being presented in the EPA studies of environmental tobacco smoke, showing in general relatively weak effects and data that is contradictory and may be hard to interpret by the lay reading. Yet, Dr. Batta Gori is willing to accept a handful of studies showing these effects where he is rejecting the evidence that is presented in this document of more than 35 studies suggesting that there is a positive association between environmental tobacco smoke and lung cancer.

I think it is an indication of the selective and biased way that the tobacco industry and their advocates have tried to look at this data, without putting it into a structure or a quantitative analysis of what the net effect and how you could combine the evidence in a quantitative sense, as was done in the EPA study using specific rules in what we call a metanalysis.

To me, this is an important advance, it's an important development on the metanalysis that was done by the National Academy of Sciences in 1986, by the Surgeon General's report in 1986, and this is just an update of those studies using the same techniques and more effectively using those techniques in this current review.

I was also very interested in Mr. Robertson's testimony. I think he made some very good points here that we have some very important indoor air pollution issues that have to be dealt with in this country. I do take issue with him though that environmental tobacco smoke is simply another indoor air pollutant that we have to deal with. In many cases, and certainly for many of the issues of sick buildings and so forth, the issue is poor ventilation and inadequate maintenance, exactly the issues he raised there. There probably are some success stories.

I just wanted to point out he mentioned the case of the Salt Lake City Airport as opposed to the Dallas Airport and the problems

that were encountered in Dallas with littering, congestion and the public image associated with putting smokers outside. I would suggest that is a very minor compared to the potential for cancer or other respiratory disease among the patrons or the workers in that facility.

Although it might be true, and I do believe they have had a real success in the Salt Lake City Airport in controlling environmental tobacco smoke, this is also the area of the country where they have the lowest smoking rates of any place in the country. This is where you have the Church of Latter Day Saints and smoking rates are less than 10 percent among that population. It's a much cleaner background that you have to work with compared to Dallas, for example, or Washington, D.C.

Mr. Chairman, you raised the question about other sources of indoor air pollution and there certainly are important sources in the indoor environment—bioallergins, fungi, bacteria and so forth. One of the prime examples we know about is legionella breeding in the ventilation systems and being transported in and being the source of a very serious and deadly disease.

The solution to that problem, when you identify a legionella outbreak, a source of legionella in a hospital or other public health facility, is not to improve the ventilation of that, it's to remove the source.

You talked about asbestos. There's a lot of debate now about what should be done about asbestos in the indoor environment. I happen to believe that removing asbestos is probably not the most efficient way to control this problem. As was noted by Mr. Robertson, you're probably releasing more asbestos into the air than you would have if you'd just left it in place and encapsulated it and brought it under control.

The issue is, if you have a source of asbestos and if you have asbestos in the walls here it's really not a source; it's only when it starts flaking off when it becomes friable, when you have an identifiable source, you're not going to control that by increasing ventilation. You want to remove that source; you want to get the asbestos out of there or encapsulate it or something like that. It's not a question of ventilation. If you have a known carcinogen in your building and you have a continuing source of this, you want to get it out of the building.

In this case, I think we have the similar example with environmental tobacco smoke. Tobacco smoke has been shown, in any way you want to look at this—in animal studies, in in vitro studies, in the laboratory, in epidemiology studies—it has been consistently shown to be a carcinogen, a very strong carcinogen. To be exposing people to a carcinogen in the indoor environment is just not acceptable. The way to control this is to take it out of the environment. I think to suggest that we should do this by increasing the ventilation rates is really not an acceptable solution to this problem. This is a very serious health hazard that we have to face up to.

I think we should not be fooled by trying to look at difficult or presumably obtuse issues regarding statistics, for example, or how you evaluate these studies, when the key issue is we know tobacco smoke is a carcinogen, we have to remove it from Federal buildings

and all other public buildings. I strongly support this bill that is being proposed.

Mr. TRAFICANT. Thank you, Doctor.

Before Chairman Valentine has a chance to interact with you, I will yield to Mr. Clyburn.

Mr. CLYBURN. I yield to Mr. Valentine.

Mr. TRAFICANT. Mr. Valentine?

Mr. VALENTINE. Thank you, Mr. Chairman and Mr. Clyburn.

Doctor, I wanted to ask you some questions about the EPA report. First of all, you may or may not know that, I believe perhaps you do know, several months before the EPA released the final version of the ETS risk assessment, a very large ETS study was published by the American Journal of Public Health. This study, I'm told, sponsored in part by the National Cancer Institute, is the largest study of its kind ever conducted in the United States and, as I understand it, is of a design similar to other studies EPA considered.

As I understand it, when that study and another recent U.S. study are added to EPA metanalysis, the resulting risk assessment is not statistically significant, even if the lower 90 percent confidence interval is applied.

The question is, do you think that it was appropriate for EPA to simply ignore this study, even though it's quite large and clearly relevant to EPA's analysis just because it would have affected EPA's result?

Dr. DOCKERY. Clearly not. The issue is that if you're going to do a review of the literature, you want to be as comprehensive as you possibly can and selection of studies based on the results of the studies is clearly inappropriate.

I'm a little confused about the specific study here. There were two studies mentioned this morning, the Stockwell and the Bronson study. Was this one of those that you're referring to? Could you clarify that for me?

Mr. VALENTINE. I can't clarify it. Is there anybody else here who can?

Dr. DOCKERY. I will say I had the chance to look at the two studies that were mentioned this morning—the Stockwell and the Bronson study—and actually neither one of those qualified as being the largest study that has been done so far. I guess the Bronson study had been published in the American Journal of Public Health and the Stockwell study in the Journal of the National Cancer Institute.

In either event, in my quick evaluation in putting those into the metanalysis, which I have on a spreadsheet on my computer back in my office, I couldn't see they had the effect that you suggested of moving the net result to a null association here. My evaluation of those two particular studies was in fact they were at very wide confidence intervals and therefore, had very little influence regarding the other studies.

Mr. VALENTINE. Let me say that my reason for asking these questions is I don't really individually have any problem with considering an individual's judgment. Smoke, whether you ingest it into your system or whether somebody else blows it to you, you can see that. The problem I have and the thing that concerns some of

us are things like what we understand to be the EPA's Science Advisory Board acknowledgement that ETS could not be classified as a Group A carcinogen based on EPA's guidelines for that type of assessment.

There again, if I misunderstand, then please clarify. I understand that EPA's Science Advisory Board and furthermore, that the ETS assessment diverges from the guidelines in several respects. This is so important because people say, look, the EPA has made a scientific study and they have made these conclusions. It concerns some of us that EPA classified ETS as a Class A carcinogen based primarily on its similarity to active smoking which is the first time that we understand it in the history of EPA that a substance has been so classified because of the fact that it looks like something else.

Second, the risk assessment in the EPA report is quite small and because there are no actual data on exposure, it's difficult to rule out the possibility it's something else, and yet, the SAB dismissed the fact that the ETS fails to meet the agency's guidelines, suggesting that if ETS could not be classified as Group A according to guidelines, then the guidelines and not the conclusions should be changed.

Say again, your judgment or my judgment as to whether somebody smoking in this room is going to affect you—but does it disturb you at all as a scientist that the EPA very blatantly changed the rules that have governed all of its other scientific enterprises just in the case of environmental tobacco smoke?

Dr. DOCKERY. Mr. Valentine, I'm not sure I can comment knowingly about this issue in that these are some issues regarding definitions and legal definitions of what a carcinogen is. I am not familiar with the deliberations that went on by the Science Advisory Board. If you're willing to let me speak realizing that I might not be expert in this area.

Mr. VALENTINE. I thought you were.

Dr. DOCKERY. I'm not. I consider myself an expert here in epidemiology and the basic science, but not in the policy decisions that are being made by EPA.

Mr. VALENTINE. I'm not talking about a policy decision, I'm talking about this so-called, scientific report. Are you familiar with it?

Dr. DOCKERY. I've read this in a lot of detail. You were talking about the Science Advisory Board evaluation of this report?

Mr. VALENTINE. I'm talking about the report itself.

Dr. DOCKERY. In my evaluation, the report makes a very strong case that environmental tobacco smoke is a carcinogen.

Mr. VALENTINE. I don't think the question is whether it makes a strong case in your judgment. I'm not a scientist and I don't know all how you refer to this, but there is a thing called a 95 percent confidence that they use and they brushed that aside and issued this report based on a 90 percent, didn't they?

Dr. DOCKERY. Let me say this about the confidence intervals. As I said, I'm a practicing epidemiologist; I consider myself a seat of the pants epidemiologist and to me I do not look to statistical significance or confidence intervals as being the hallmark of defining what a health effect is. This is a point that I pound into my students from the first day of class until the last day of class, that the

statistical significance, as quoted from me, is a measure of the stability and how much confidence we have in that in a particular estimate of the risk in this case of the association between husbands smoking and lung cancer incidence in their wives.

Mr. VALENTINE. Dr. Dockery, I understand that feeling but are you here to tell us that your testimony is based on your conclusions arrived at otherwise besides from this report?

Dr. DOCKERY. I'm not sure I follow the question. I will say that I think this report stands not only the statistical significance test that is presented there.

Mr. VALENTINE. How can you have a scientific report that has certain criteria, certain standards you live up to—let me say parenthetically that according to our information, there are more individual studies in the case of EMF, 40 percent for EMF, as opposed to 30 or so for ETS. They generally report higher, relative risks, yet EPA called the results for EMF far from conclusive. Similarly, I'm told that scientists who evaluated the data on chlorinated water say that the relative risks are stronger than ETS.

I say again, I'm not talking about individual concepts but the EPA has presented a report and when they got it together, they changed the rules in the middle, didn't they?

Dr. DOCKERY. That's not the way I see it at all.

Mr. VALENTINE. How do you see it?

Dr. DOCKERY. You made the statement they defined environmental tobacco smoke as being a carcinogen because of its similarity to tobacco smoke. That, I see, as the crux of the argument. We know tobacco smoke is a carcinogen. It's been shown in many, many studies. Why should we believe that it is not a carcinogen when applied at low doses to nonsmoking people?

We have many examples where this has been applied in asbestos exposure. There is effectively no data that asbestos exposure, in nonoccupationally exposed people, causes increased mesothelioma or other lung cancers. Yet, we've regulated asbestos based on extrapolation of the high dose exposures.

Here, we're simply extrapolating from the high dose exposures for active tobacco smoke down to where we think the evidence is for environmental tobacco smoke.

Mr. VALENTINE. Let me ask you this. Didn't EPA change the rules from the 95 percent to the 90 percent?

Dr. DOCKERY. I can't say. I really don't know the answer to that. Whether they based their decision on a 95 percent or 90 percent confidence interval, in my own evaluation of these data, taking these and doing an independent evaluation, I certainly find a statistically significant association when you do a metanalysis between environmental tobacco smoke and lung cancer, even including the two latest studies that have been added since the publication of this document. That would be based on 95 percent confidence intervals with all the studies here, plus the recent studies that have been added.

Mr. VALENTINE. What data are you talking about?

Dr. DOCKERY. I'm talking about all the data that is presented in the EPA document that you're referring to.

Mr. VALENTINE. You went back and restudied the questions and answers they received and all of the scientific material?

Dr. DOCKERY. I've gone back and looked at those articles and extracted the key results of those and done a metanalysis. A metanalysis is not some magic blackbox here. It's a very simple technique that is routinely now applied in epidemiology.

Mr. VALENTINE. Have you studied other reports put out by the Environmental Protection Agency?

Dr. DOCKERY. I've studied most recently the criteria document for nitrogen dioxide where metanalysis was done to look at the effects of nitrogen dioxide on indoor nitrogen dioxide associated with gas stoves on respiratory health of kids and again, I think that was an exemplary study.

Mr. VALENTINE. What about the electromagnetic radiation, EMF?

Dr. DOCKERY. I've had the chance to look at that literature also. I personally think it's a little ambiguous right now but I am convinced that there is an association there with some measure of electromagnetic fields. I don't think we know what the key exposure is in those particular studies, whether it is the magnetic fields, the electric fields. There is something going on there with wiring configurations and electromagnetic radiation but it's not clear at this point what the key exposure is. It's very different from environmental tobacco smoke where we have a well-known marker, a well-known cause of cancer that we can specifically identify as being the exposure in people who are participating in these epidemiologic studies.

In the EMS studies, there is a hypothesis that's been put forward but the exposure has not been identified yet.

Mr. VALENTINE. You're giving your opinion, but the EPA report, they did change the rules, didn't they?

Dr. DOCKERY. I'm not sure I can answer that, sir.

Mr. VALENTINE. You've looked at it, you've studied it. Who could be better able to look at it than you? Why do you hesitate? You know they changed the rules.

Dr. DOCKERY. No. I do not consider a 95 percent confidence interval a point value of 5 percent error, which is what you're implying, as an acceptable definition of an adverse health effect. That is outdated epidemiology at this point and is no longer accepted in modern epidemiologic practice. That dates back to hypothesis testing that was developed for laboratory experiments in agriculture and in physics and other settings like that to evaluate the effect of random errors in the data.

In epidemiology, we are not experimenting on people; we are doing observational studies. We are not experimenting; we do not have control over all the variables in the study and there is a growing body, and I think the overwhelming body of scientific thought in epidemiology right now is that placing an absolute red line at a 5 percent statistical significance level when you have all of the studies here effectively all but one study that we've talked about either this morning or the 30-some studies that are presented by the EPA are consistent with an adverse health effect with increased cancer associated with spousal smoking, all but one.

Mr. TRAFICANT. Would the gentleman yield briefly for one question?

Mr. VALENTINE. Yes.

Mr. TRAFICANT. That one, is that the Bronson study?

Dr. DOCKERY. No. In fact, the Bronson study shows a positive effect. It's a small relative risk, it's 1.03, I think, but in fact, it's consistent with the overall metanalysis.

The Stockwell study, which was mentioned, had a relative risk of 3. It turns out to be highest estimated risk of any study that's been published so far.

Mr. TRAFICANT. Isn't it a fact that the Stockwell and Bronson studies both, when looked in conclusive forms, do in fact conclude that they suggest a small but consistent increased risk of lung cancer from passive smoking?

Dr. DOCKERY. Indeed. That's my reading of those studies.

Mr. TRAFICANT. I yield back to the gentleman.

Mr. VALENTINE. I want to thank the Chairman for being very considerate and very generous.

I would like to ask, if I can, the gentleman to submit the studies.

Mr. TRAFICANT. Mr. Clyburn?

Mr. CLYBURN. None.

Mr. TRAFICANT. I'd like to also read one question brought forward by Mr. Valentine before he leaves. EPA loosens its judgment criteria specifically for the risk assessment from 95 to 90 confidence intervals. Without this loosening, is the criticism, results would not be statistically significant. The 90 percent confidence interval is consistent with the one tail significant test which was used in both draft reports as well as in the final report and which is fully appropriate for this assessment given the known positive association between active smoking and lung cancer.

Furthermore, the conclusions are not dependent on the criterion. Several of the analyses conducted demonstrate with much greater than even 95 percent certainty that the full set of results did not occur by chance.

We have gotten into splitting some hairs here. You've been questioned very extensively, Dr. Dockery, but for the record, you're saying that from your professional position, including your professional discipline and training, your review of the scientific documents admitted now for EPA which supports previous findings from the Surgeon General, that ETS is in fact a known carcinogen in your opinion and is, in fact, dangerous to the health of Americans exposed to it?

Dr. DOCKERY. Yes, sir.

Mr. TRAFICANT. You believe it is almost not impossible to remove some of these elements from our buildings. If I were to assume you said this, you'd be far better off seeing all the asbestos placed in some type of a protective device so they could never be airborne because only when they are airborne do they affect our health. Is that correct?

Dr. DOCKERY. That's right. What's required there is total encapsulation of the asbestos.

Mr. TRAFICANT. Is there any way we can totally encapsulate the passive smoke from a smoker in a building?

Dr. DOCKERY. I'm sure the engineers and Mr. Robertson can come up with a fix for this somehow.

Mr. TRAFICANT. Would that be the mission of the new space station or just a Star Trek rerun? In all honesty, you feel very strong-

ly that the EPA's statement and studies and number of studies are a good parameter for advice to the Congress of the United States?

Dr. DOCKERY. I think it is a very strong, scientific document that can be used for basing those decisions on. With regard to control of indoor air pollution, I think the issues we see as were alluded to by Mr. Robertson, are really not generally in the design of adequate systems, but in the breakdown of the maintenance and the operation of those systems. I'm sure you can design the optimum system that could remove the smoke in some sense from an individual smoker, but in fact people violate the rules and there are changes and each generation of new engineers coming into a building has to learn all the lessons his company has had to teach.

Every building you go into, you see these systems breakdown. We cannot really rely on engineering fixes when we have a very seriously potential carcinogen in the indoor environment.

[Subsequent to the hearing, additional questions were submitted to Professor Dockery by Representative Valentine. The questions and responses follows:]

HARVARD SCHOOL OF PUBLIC HEALTH,
Boston, MA, July 19, 1993.

SUSAN F. BRITA,
Staff Director, Subcommittee on Public Buildings & Grounds, House of Representatives, Washington, DC.

DEAR MS. BRITA: Following my testimony before the subcommittee on April 22, 1993, I received two sets of questions from Representative Valentine. I am enclosing a copy of those questions plus my response. I trust you will find this material useful and will distribute it as appropriate.

Sincerely yours,

DOUGLAS W. DOCKERY,
Associate Professor, Environmental Epidemiology.

HEARING ON H.R. 881—QUESTIONS FROM REPRESENTATIVE VALENTINE

ON EPA'S USE OF A 90 PERCENT CONFIDENCE INTERVAL

1. Q: Do you agree with EPA that it was appropriate to switch from 95 percent to 90 percent confidence intervals in the ETS report, even though 95 percent is the universally accepted scientific standard?

A: Use of a 90% (rather than 95%) confidence interval in this case corresponds to use of a one-tailed (rather than two-tailed) test of statistical significance at the p=0.05 level. In the case of a known carcinogenic agent, such as tobacco smoke, it is entirely appropriate to use a one-tailed or one-sided test of significance for an adverse effect of tobacco smoke. We would not expect tobacco smoke to be a protective agent against cancer and therefore the use of a 5% one-tailed test, or equivalently a 90% confidence interval, is justified.

2. Q: I'm told that in the great majority of the individual studies EPA evaluated, in the first draft of the ETS risk assessment and in virtually all other EPA risk assessments, the Agency has relied upon 95 percent confidence intervals. Yet in the final draft of the ETS assessment, EPA had switched to a 90 percent confidence interval. And, as we discover, the studies considered in that final draft didn't produce a statistically significant result at the 95 percent level. Is it your view that all of that is simply confidence?

A: The confidence interval reflects only one of several elements that must be considered in the evaluation of epidemiologic studies. The confidence interval is a quantitative measure of the statistical or random error in the data and reflects, therefore, only the influence of chance in these data. It is inappropriate to define an adverse health effect based on a statistical significance or a confidence interval, and therefore whether this is based on the 90% confidence level or the 95% confidence level is irrelevant.

3. Q: You've done a considerable amount of original research yourself, I believe. Have you ever reported your results in terms of 90 percent confidence intervals? If not, why not, and if so, what was the basis for your decision?

A: The purpose of the confidence interval is to estimate the statistical variance, that is, the confidence in the effect being estimated. Causality is not determined by crossing an arbitrary statistical line. Statistical significance is only one of many criteria that must be used in evaluating cause and effect. In many cases we have reported harmful or beneficial effects which do not achieve statistical significance based either on a 5% two-tailed or on a 5% one-tailed test, that is, on bounding the so-called no-effect estimate, or null hypothesis.

ON META ANALYSIS

4. Q: EPA considered 11 U.S. studies in its meta analysis of the ETS data. I am told that not one of those 11 studies originally reported a statistically significant overall increase in risk at the 95 percent confidence level. Only by combining the studies, adjusting the data and lowering the standard of confidence could EPA claim that the result was statistically significant. Do you consider it valid to take 11 studies which show nothing individually and artificially combine them to produce a significant result?

A: Meta-analysis is a significant innovation in the review of scientific literature. It provides an objective method for combining data and evaluating the effects estimates from various studies. Moreover, it provides a method for contrasting various studies that are in the literature and for evaluating the effects of bias, confounding, and other important issues in epidemiological studies. In this sense, I think that the EPA analysis, in following similar meta-analyses of ETS and lung cancer by the National Academy of Sciences[1] and the U.S. Surgeon General,[2] has been very responsible in applying this technique to an environmental agent. I strongly support the use of meta-analysis in rule-making.

5. Q: Do you think 11 studies which essentially show nothing are an adequate basis for a Group A classification?

A: I disagree with your assertion that these studies show nothing. While most of these studies fail to achieve statistical significance, they are universally consistent with a positive association between environmental tobacco smoke and increased risk of lung cancer. Therefore bringing these studies together in the meta-analysis is an appropriate evaluation of their strengths. But these studies have to be evaluated based on other potential sources of error, confounding, and bias, as well as random error which is measured by the confidence interval. I think the EPA has done an excellent job in evaluating these alternative explanations, both in the meta-analysis and in their qualitative review.

ON EPA'S OMISSION OF DATA

6. Q: You may or may not know that several months before EPA released the final version of the ETS risk assessment, a very large ETS study was published in the American Journal of Public Health. This study, sponsored in part by the National Cancer Institute, is the largest study of its kind ever conducted in the U.S. and, I'm told, is of a design similar to the other studies EPA considered.

As I understand it, when that study and another recent U.S. study are added to EPA's meta analysis, the resulting risk estimate is not statistically significant even if the lower 90 percent confidence interval is applied.

Do you think it was appropriate for EPA to simply ignore this study even though it is quite large and clearly relevant to EPA's analysis, just because it would have affected EPA's result?

A: When a document such as the EPA report is being prepared, there is obviously some cutoff date at which the work and the editing stop and the document goes to publication. With new studies coming out continually showing the adverse effects of environmental tobacco smoke, there comes some point, largely determined by forces outside the control of the reviewers, when the writing has to stop. The EPA report has a publication date of December 1992. The study by Brownson et al.[3] in the November-1992 issue of the American Journal of Public Health (which means it arrived in my office in late November or early December). The Stockwell et al

[1] National Research Council. (1986) Environmental tobacco smoke: Measuring exposures and assessing health effects. Washington, DC: National Academy Press.

[2] U.S. Department of Health and Human Services. (1986) The health consequences of involuntary smoking. A report of the Surgeon General. DHHS Pub. No. (PHS) 87–8398. U.S. Department of Health and Human Services, Public Health Service, Office of the Assistant Secretary for Health, Office of Smoking and Health.

[3] Brownson RC, et al. (1992) Passive; smoking and lung cancer. American Journal of Public Health 82: 1525–1530.

paper[4] appeared in the September issue of the Journal of the National Cancer Institute. Given the dates, one can hardly suggest that these papers were ignored because they may have "affected EPA's results." Moreover, as discussed later, inclusion of these papers would not affect the meta-analysis.

ON THE EPA GUIDELINES FOR CARCINOGEN ASSESSMENT

7. Q: I am told that even EPA's Science Advisory Board acknowledged that ETS could not be classified as a Group A carcinogen based on EPA's guidelines for Carcinogen Assessment. I understand that the ETS assessment diverges from the guidelines in several respects. First, EPA classified ETS as a Group A carcinogen based primarily on its similarity to active smoking, which is the first time in EPA history that a subsection has been so classified based on the claim that it looks kind of like another substance. Second, the risk estimate in the EPA report is quite small, and because there are no actual data on exposure, it is difficult to rule out the possibility that something else accounts for the observed risk.

And yet, the SAB dismissed the fact that the ETS fails to meet the Agency's guidelines, suggesting that if ETS could not be classified as Group A according to the guidelines, then the guidelines—and not the ETS conclusions—need to be changed.

Does it disturb you at all, as a scientist, that EPA very blatantly changed the rules that have governed all of its other scientific enterprises just in the case of ETS?

A: I was not part of the EPA Science Advisory Board deliberations and therefore cannot comment on their reasoning. I agree with and endorse their conclusions. You suggest that ETS has been classified as a Group A carcinogen based on its similarity to active smoking. I would agree with such a line of reasoning. The same chemical agents, including many known carcinogens, which are found in main-stream tobacco smoke are also seen in side-stream tobacco smoke, sometimes at higher concentrations. Based solely on the presence of these carcinogens in side-stream tobacco smoke, I think it is appropriate to classify environmental tobacco smoke as a carcinogen. We have many similar examples, such as coke-oven emissions and asbestos, where effects have been demonstrated in occupational or high-exposure areas and which have led to regulation of those same emissions as environmental carcinogens. Secondly, you suggest that the risk is small, or at least compared to the risk from active smoking. But that does not mean it is not important. In fact, compared to the relative effect that would be expected, given the quantification of exposure from environmental tobacco smoke compared to active tobacco smoke, the effect is much larger that one would expect if the effect were scaled by the relative exposures. Moreover, because of the large number of people exposed, the risks of cancer from ETS imply many more incident cancers than would be expected from coke-oven emissions, absestos, or other Class A carcinogens. Therefore, I hardly consider this a change in the rules and find it consistent with previous practice.

8. Q: What other substances can you identify for which it would be appropriate to change the rules if the rules don't produce the result you want?

A: As described above, this rule-making has followed the same rules which have generally been applied and I do not accept your suggestion that rules have been changed for environmental tobacco smoke.

9. Q: If EPA's guidelines for Carcinogen Risk Assessment shouldn't be relied upon to produce the Agency's risk assessments, then what rules should govern the risk assessment process? Should all federal agencies simply abandon risk assessment principles, and do whatever seems to make the most sense to them?

A: This evaluation of the effects of environmental tobacco smoke has brought a high degree of quantification and scientific rigor to the field of risk assessment which in the past was based on individual evaluations of epidemiologic and toxicologic studies. The combining of data in the meta-analysis and the quantitative and rigorous evaluation that is applied has set an important precedent for future studies.

ON THE PRECEDENTS SET BY THE ETS RISK ASSESSMENT

10. Q: You may or may not be aware of comparisons that have been made between the strength of the data on ETS and the strength of the data on electromagnetic radiation (EMF), for example, and chlorinated water. According to my information, there are more individual studies in the case of EMF—40 for EMF as opposed to 30 or so for ETS—and they generally report higher relative risks. Yet

[4] Stockwell HG, et al. (1992) Environmental tobacco smoke and lung cancer risk in nonsmoking women. Journal of the National Cancer Institute 84: 1417–1422.

EPA called the results for EMF "far from conclusive." Similarly, I'm told that scientists who have evaluated the data on chlorinated water say the relative risks are stronger than for ETS.

If EPA conducted a meta analysis on the data for these substances, applied a 90 percent confidence interval and produced a statistically significant result, would you agree that they should be classified as Group A carcinogens?

A: The important difference is that there is little or no supporting evidence for the EMF hypothesis available in the literature at the moment. The meta-analysis is only one component of a multi-factorial evaluation that has to be undertaken in classifying an agent as a carcinogen. While evidence for a positive association may be found for EMF, there is a lack of evidence for strong occupational effect or evidence of other effects at high-EMF exposures as is shown for direct tobacco smoke. And secondly, there is a lack of strong toxicologic basis that has been developed for tobacco smoke.

11. Q: Why do you think EPA finds the ETS data so much easier to interpret than it finds the data for EMF or chlorinated water?

A: Tobacco smoke has been shown to be a carcinogen in laboratory experiments; tobacco smoke has been shown to be a carcinogen in studies of humans actively smoking themselves, and, in multiple studies, environmental tobacco smoke has been shown to be an environmental carcinogen. The difference for EMF or chlorinated water is that there is not the same body of evidence suggesting that there are effects being demonstrated in laboratory experiments or at very high exposures, and until such a body of evidence is developed, then it would be inappropriate to move forward with a similar finding for those particular agents.

12. Q: Assume, for example, that EPA found that the smoke produced by burning wood in your fireplace was similar to coke-oven emissions, which EPA believes to be a Group A carcinogen, in terms of their physical and chemical make up. Would you think that would be reason enough to label diesel exhaust a Group A carcinogen?

Then do you agree with the statement in the ETS risk assessment that the similarity between ETS and active smoking are enough to justify a Group A ranking for ETS even without any further studies?

A: The question is not whether these chemicals are similar for the same specific carcinogenic chemicals are present in main-stream and side-stream smoke. When people are exposed environmentally to the chemical species that has been shown in the laboratory or occupational settings to be carcinogenic, and when epidemiologic studies show increased cancer risk from environmental exposure, then there is clear evidence for a Class A finding. Coke-over emissions have clearly been shown to be strongly carcinogenic among workers; specific agents from coke-oven emission have been demonstrated to be carcinogenic in laboratory experiments. If wood smoke were shown to contain some of those same chemicals, it would raise the question of whether they were carcinogenic. Then the second question that would have to be addressed is whether those effects can be extrapolated to environmental exposures, and it would be the job of the epidemiologists to evaluate those effects in a realistic setting.

13. Q: Are you, as a scientist, comfortable with the principle of basing Group A classifications on similarities between substances? What about basing them on very small risk estimates when you cannot be sure that all important factors have been considered?

A: As implied before, declaring a specific chemical to be a Class A carcinogen should be based, on the combination, first, of evidence from high-exposure evaluations in human populations that there are effects; second of supporting pathologic or physiologic evidence from laboratory experiments, and third, of evidence from epidemiologic studies suggesting that, at characteristic environmental exposures, excess risks can be observed whether or not they are statistically significant.

In the case of environmental tobacco smoke, the evidence is very clear on all three counts. Tobacco smoke has been shown to be a carcinogen among active smokers and we would expect that small risks would also be shown among nonsmokers passively exposed to tobacco smoke. The quantitative risk estimates found in epidemiologic studies are consistent with measured exposures in nonsmokers and provide quantitative evidence for those associations. The EPA document evaluated the important alternative explanations that have been proposed by the tobacco interests and has shown, to my satisfaction, that these cannot explain the observed associations between ETS and lung cancer. There is no evidence in the data to suggest that there are uncontrolled confounders of sufficient magnitude to create the observed associations that have been found in the epidemiologic studies.

ON CREDIBLE SCIENCE, CREDIBLE DECISIONS

14. Q: A panel of scientific experts who were asked by former EPA Administrator Reilly to review the quality of science at EPA issued a report last year entitled Safeguarding the Future; Credible Science Decisions. The report was quite severe in its criticisms of EPA science. To cite just two of the conclusions, the report said EPA science is "of uneven quality" and is "frequently perceived" to be "adjusted to fit policy." That certainly sounds like the quote from the scientist critical of the ETS risk assessment, who said that "The science of which EPA avails itself is that which happens to fit the political agenda of the moment."

Do you agree that EPA tends to exaggerate risks, or "adjust" science, on its preferred policies or on what is politically correct?

A: The EPA review of the health risk associated with ETS is a good example of how risk assessment should be done, using objective criteria for assessing risk and quantitative methods for evaluating the potential for bias and confounding in the epidemiologic studies. This contrasts with the subjective measures which have been used in the past and which are subject to political bias rather than scientific objectivity.

15. Q: How do you view the recent Alar scare, or the apparent exaggeration of dioxin risk, which seem to reflect the same problem at EPA?

A: The EPA analysis of the health effects of tobacco smoke reflects an objection evaluation of a vast array of direct studies—of smokers heavily exposed to smoke, of laboratory experiments, of animal experiments, of epidemiologic studies of the environmentally exposed subjects—all of which point to environmental tobacco smoke as a carcinogen. This vast body of evidence is certainly more compelling than the evidence that was presented in some of the other decisions that have been made by the EPA recently.

QUESTIONS FOR PROFESSOR DOCKERY

1. Q: You were invited by the Subcommittee to give testimony. Please provide the amount and source of any financial support provided to you in connection with your appearance before the Subcommittee.

A: Indeed, I was invited by the Subcommittee to give testimony. I have received no financial support for my time. I have recently been reimbursed by the Coalition On Smoking Or Health for my travel expenses which consisted of economy airline fare, parking for the day at the airport in Boston, and my public transportation costs in Washington, DC. I appreciate your concern for my financial support, and will let you know if I need help.

2. Q: You testified that, according to calculations that you had personally performed, adding the results of two recent ETS studies (i.e., the Brownson and Stockwell studies) to the meta analysis pool used by EPA to derive a relative risk estimate for the U.S. population did not make any real difference. In particular, you said that the combined relative risk estimate remained statistically significant at both the 90% and 95% levels of confidence.

Please provide an explanation of your calculations, showing precisely what data you employed from which studies, as well as any adjustments that you made to the data. Also, if you have not already done so, please calculate the combined relative risk figure for the total number of U.S. studies (i.e., those used by EPA plus Brownson and Stockwell), using exactly the same data components and adjustments that EPA used in its risk assessment.

A: Attached is a copy of the results of my own simple meta-analysis of the original EPA data, taking the weighted mean of the studies as presented in Table 2 of the EPA report. The combined estimate over all the studies based on the crude data presented gives me an overall effect estimate of 1.24 (with a 95% confidence internal of 1.13–1.35). Including the Brownson[3] and the Stockwell[4] studies in that analysis (as shown in the attached table) only minimally changes the overall effect estimate to 1.21 (with a 95% confidence interval of 1.11–1.31).

3. Q: Are you aware of any carcinogen classifications or regulatory actions by any agency of the U.S. government in which a chemical compound was determined to be carcinogenic in humans on the basis of an epidemiologic data base that was statistically non-significant?

A: As stated earlier, statistical significance is not a measure of effect. It is simply a measure of statistical power of study size. The consistency of the epidemiologic studies reviewed in the EPA document showing a positive association between spouse smoking and lung cancer is the most compelling evidence here. The fact that most studies do not attain statistical significance at the 5% level merely indicates that these studies are not large enough to detect the effects or, alternatively, that

the crude measure of spouse smoking is not sufficiently precise to provide a statistically significant association.

4. Q: What confidence intervals, 90% or 95%, were reported in the epidemiologic studies reviewed by EPA in its risk assessment?

A: A knowledgeable or even minimally trained statistician or epidemiologist would be able to interpret either the 90% or 95% confidence interval and be able to convert from one to the other with very little effort.

5. Q: What confidence interval was employed in EPA's report of the pooled relative risk estimate in the first draft of its risk assessment in 1990? Did you review either that document or any later drafts at the request of EPA? Please provide a copy of any communications between yourself and EPA staff relating to such reviews. Please highlight any discussion of appropriate statistical methodology, including the choice of the proper confidence interval.

A: All my correspondence with EPA are in the public record. I was not asked to review the lung cancer results nor comment on the statistical methodologies. Nevertheless, I have found the statistical methods applied by EPA to be appropriate and proper.

6. Q: You testified that you disagreed with the assertion that the Brownson study was the largest study of its kind (i.e., a case-control study) conducted in the U.S. Please name any published study or studies that you consider to be larger, and explain your conclusion.

A: I consider the Fontham et al[6] study to be the best study that has been conducted and certainly the statistically most powerful study conducted so far. That is indicated by the weights given to this study in my attached meta-analysis. It has the strength of being a prospective study looking at 420 incident primary lung cancers that are pathologically confirmed with very good exposure estimates and very good definition of the health outcomes. The Brownson study that you refer to is certainly a large study, having a large number of cases (432 lifetime non-smokers). It also has a high weight given to it in the meta-analysis and, as the authors conclude in their study, "Ours and other recent studies suggest a small but consistent increased risk of lung cancer from passive smoking." This certainly is consistent with all the evidence that has been presented in the other studies.

7. Q: You testified that the similarity between mainstream smoke and ETS justifies the conclusion that ETS is a carcinogen. Are there any other substances that any agency of the U.S. government has determined to be carcinogenic because they are "similar" to some other substance?

A: I would say that this is a very consistent argument, that the same carcinogenic species that are found in mainstream smoke are found in environmental tobacco smoke and this in itself is sufficient evidence for concluding that environmental tobacco smoke is a carcinogen. This has clearly been shown for other substances declared carcinogens (asbestos, benzene, coke oven emissions, for example).

8. Q: In response to questions from Rep. Emerson, Dr. Maurice LeVois submitted a written comment to this Subcommittee on EPA's use of statistics. Please refer to pages 2–4 of his response, in which he quotes from a number of recognized authorities on statistical methodology. It is clear from these authorities, is it not, the EPA departed from accepted practice in its choice of a "one-tailed test" or statistical significance?

A: I'm sorry, I do not have Dr. LeVois's comments available to me, but I certainly do not find anything unacceptable in the use of a one-tailed test of significance in the EPA evaluation. As I've stated before, the tests of significance certainly do not indicate whether there is a health effect or not. The one-tailed test of significance is certainly appropriate here, given that there is no evidence that tobacco smoke should provide a protective effect from lung cancer.

9. Q: You testified before the Subcommittee that you believe there is a misplaced emphasis in scientific research on "a red line" drawn at 95%. Since 1986, you have been listed as primary or co-author of at least 14 research publications of which I am aware in which your results have been expressed as relative risks or odds ratios. In how many of these did you report your results at a confidence level less than 95%?

A: In all of our recent papers, we have reported the confidence intervals of our relative risk or odds ratios in terms of the 95% confidence interval. The important point here is that many of those have bounded the no-effect level of 1, that is, we have not defined effects based on statistical significance (that is, based on a p-value less than 0.5), and some of these are much greater than a p-value of 0.5. But the 95% confidence interval or the 90% confidence interval or any other arbitrary value

[6] Fontham, et al. "Lung cancer in non-smoking women: A multi-center case control study" published (1991) in Cancer Epidemiology: Biomarkers and Prevention (Volume 1, pages 35–43).

that one would ascribe to these associations provides an estimate of the confidence that you have in the point estimate.

ENVIRONMENTAL TOBACCO SMOKE: MEASURING EXPOSURES AND ASSESSING HEALTH EFFECTS—SUMMARY OF EPIDEMIOLOGIC STUDIES OF RISK BASED ON EXPOSURE ASSESSED BY SPOUSE SMOKING HABITS WHEN AVAILABLE, OR SMOKING BY THE HOUSEHOLD COHABITANTS

[Data from Table 5–5 (US EPA, 1992)]

Authors	Year	OR	90 percent CI		Var (lnOR)	Weights	ln(OR) Weight [1]
			Lower	Upper			
Case-control studies:							
Akiba et al	1986	1.52	0.96	2.41	0.078	12.78	5.35
Brownson et al	1987	1.52	.49	4.79	.480	2.08	.87
Buffer et al	1984	.81	.39	1.66	.194	5.16	−1.09
Chan and Fung	1982	.75	.48	1.19	.076	13.13	−3.78
Correa et al	1983	2.07	.94	4.52	.228	4.39	3.19
Fontham et al	1991	1.32	1.08	1.61	.015	67.90	18.85
Gao et al	1987	1.19	.87	1.63	.036	27.46	4.78
Garfinkel et al	1985	1.31	.93	1.85	.044	22.88	6.18
Geng et al	1988	2.16	1.21	3.84	.123	8.12	6.25
Humble et al	1987	2.34	.96	5.69	.293	3.42	2.91
Inoue & Hirayama	1988	2.55	.9	7.2	.399	2.50	2.34
Janerich et al	1990	.86	.57	1.29	.062	16.23	−2.45
Kabat and Wynder	1984	.79	.30	2.04	.339	2.95	−.69
Kalandidi et al[1]	1991	1.62	.99	2.65	.063	15.85	7.65
Koo et al	1987	1.55	.98	2.44	.077	13.01	5.70
Lam	1985	2.51	1.49	4.23	.101	9.94	9.15
Lam et al	1987	1.65	1.22	2.22	.033	30.20	15.12
Lee et at	1986	1.03	.48	2.20	.214	4.67	.14
Liu et al	1991	.74	.37	1.48	.178	5.63	−1.70
Pershagen et al[1]	1987	1.28	.82	1.98	.051	19.77	4.88
Shimizu et al	1988	1.08	.7	1.68	.071	14.12	1.09
Sobue	1990	1.06	.79	1.44	.033	30.03	1.75
Svensson et al	1989	1.26	.65	2.48	.166	6.04	1.40
Trichopoulos et al	1983	2.08	1.31	3.29	.078	12.76	9.35
Wu et at.[1]	1985	1.41	.63	3.15	.169	5.93	2.04
Wu-Williams, Samet	1990	.79	.64	.98	.017	59.62	−14.05
Brownson et al[1]	1992	1.03	.80	1.20	.011	93.47	2.76
Stockwell et al[1]	1992	1.60	.80	3.00	.114	8.80	4.13
Cohort studies:							
Butler[1]	1988	2.02	.48	8.56	.540	1.85	1.30
Garfinkel[1]	1981	1.17	.85	1.61	.027	37.66	5.91
Hirayama	1984	1.38	1.03	1.87	.033	30.43	9.80
Hole et al[1]	1989	2.27	.4	12.7	.778	1.29	1.05

	ORmh	95 percent CI		Var (lnOR)	Weights	
		Lower	Upper			
Overall for case-control studies	1.23	1.11	1.35	0.205	416.57	85.22
With Brownson and Stockwell studies	1.19	1.10	1.30	.178	518.84	92.12
Overall for Cohort studies	1.29	1.02	1.63	.254	71.23	18.07
Overall for studies in EPA report	1.24	1.13	1.35	.212	487.80	103.29
With Brownson and Stockwell studies	1.21	1.11	1.31	.187	590.07	110.19

[1] 95 percent confidence intervals reported.

Mr. TRAFICANT. Thank you, Dr. Dockery.

Without further questions, this meeting stands adjourned.

[Whereupon, at 12:00 p.m., the subcommittee was adjourned, to reconvene at the call of the Chair.]

PREPARED STATEMENTS OF WITNESSES

STATEMENT OF DR. GIO BATTA GORI

Mr Chairman, Members of the Subcommittee.

I am Dr. Gio Batta Gori. I will not restate my qualifications since, as you recall, I testified at the last hearing on H.R. 881, on March 11, 1993. At that time, The Tobacco Institute had asked that I present my views as a scientist in regard to the report on environmental tobacco smoke (ETS) issued by the Environmental Protection Agency on January 7, 1993[1].

The Tobacco Institute has asked that I appear before you again to offer some necessary clarification to the testimony recently submitted for the record by Dr. Alfred Munzer, on behalf of the Coalition on Smoking OR Health.

All the same, the views expressed here are my own and not necessarily those of The Institute.

In his testimony, Dr. Munzer mentions especially two studies published before the release of the EPA report on ETS, but excluded from the report itself, claiming that they sustain the EPA's report conclusions. These are the studies of Stockwell et al.[2] and Brownson et al.[3], and a close look at their data as published leads to quite obvious conclusions, opposite to those reached by Dr. Munzer.

In epidemiologic jargon, risk is reported either as relative risk (RR) or as their close approximations, the odds ratio (OR) values. Expressed values are relative to the control group, which is assigned a value of 1. Values greater than 1 suggest increased risk and values below 1 suggest decreased risk, relative to the control group. In the abstract to their paper, Stockwell at al. write:

> "The risk of lung cancer more than doubled for women who reported 40 or more smoke years of household exposure during adulthood (odds ratio [OR] = 2.4 ...) or 22 or more smoke years of exposure during childhood and adolescence (OR = 2.4 ...)... When a surrogate respondent other than the patient's husband provided information of exposure, the risk estimates were considerably lower. Conclusions: These findings suggest that long term exposure to environmental tobacco smoke increase the risk of lung cancer in women who have never smoked."[2]

What the abstract fails to indicate is that data in the paper itself show that, when surrogate respondents other than the patient's husband provided information of exposure, the risk estimates were not only considerably lower, but were actually below 1, therefore suggesting that nonsmokers exposed to ETS had a <u>lower</u> lung cancer risk than those who were not exposed[4].

What would a scientist do when confronted with such conflicting results? First, no conclusions would be made on that basis. Second, an investigator would try to determine which source of exposure information is credible, if any. For instance, data reported in the Stockwell et al. paper inevitably pose two

questions. One is whether husbands, daughters, or sons are credible sources of information for ETS childhood exposure of their wives/mothers. The other is whether women with lung cancer might -- by the very questions posed to them -- be induced to find blame by amplifying or even imagining a childhood exposure to ETS presumably generated by their own parents or relatives[2].

Equally unresolved uncertainties are to be found in the Brownson et al. study, one of the largest on record[3]. According to the abstract of the paper:

> "No increased risk of lung cancer was associated with childhood passive smoke exposure. Adulthood analyses showed an increased lung cancer risk for lifetime nonsmokers with exposure of more than 40 pack-years from all household members (odds ratio [OR] = 1.3...) or from spouses only (OR = 1.3...)... Conclusions: Ours and other recent studies suggest a small but consistent increased risk of lung cancer from passive smoking. Comprehensive actions to limit smoking in public places and worksites are well-advised."[3]

Actually, data in this study reveal not only that childhood exposure was <u>not</u> associated with an excess risk of lung cancer, but that such exposure gives odds ratios that are substantially below 1, and thus reports a reduced risk for lung cancer in ETS exposed individuals. What should one make of these results, which are in direct conflict with the reports by Stockwell et al., as noted above? At the very least an impartial investigator would suspend judgment until further clarification becomes available.

Curiously, the abstract of the Brownson et al. paper fails to note that data reported in table 2 of the paper itself are quite at odds with the author's summary conclusions about risk from ETS. To begin with, the overall analysis of risk fails to uncover an excess of risk. A small excess risk is reported only for selected categories of highest exposure, and with any other exposure level an apparent decrease of risk is reported[6]. Since overall risk is the index used in all other studies considered by the EPA report, the Brownson et al. study clearly does not support EPA's conclusions despite what its abstract suggests.

This set of results clearly conflicts with the overall conclusions of the authors. Besides, in view of the Stockwell et al. data, should Brownson et al. worry that 74% of their exposure information comes from offspring or siblings and not from the patients themselves? And what about their conclusion that smoking restrictions in workplaces are well advised, when Stockwell et al. and most other studies failed to find an association for worksite exposures to ETS, and the Brownson et al. paper itself states in its text that "there was no elevated risk of lung cancer risk associated with passive smoke exposure in the workplace..."?

In fact, the entire literature assembled by the EPA report on ETS -- both for lung cancer and respiratory conditions -- suffers contradictions similar to the Stockwell and Brownson studies, and like these studies raises more questions than it can answer. And

I have touched here only on the superficial problems that become obvious from the reported data themselves. There are other and major fundamental problems of which I spoke in my previous testimony before you, namely how to adjust for misclassifications of exposed and unexposed individuals, for presumed background ETS exposures, and for several lung cancer and respiratory risks that are known to confound the results.

Of course, there is also the question of statistical significance. While it is true that statistical significance is a matter of consensus, it is also true that the universal consensus prescribes margins of error ±5% or lower, and frowns upon the ±10% margin that EPA insisted on using in the ETS report. In fact, EPA would certainly reject a petition for the registration of a pesticide, for instance, if it were submitted to the agency with data based on a ±10% statistical standard. Professor Dockery, whom Dr. Munzer quotes, is right when stating that the statistical significance of data "...reflects the stability, that is the uncertainty, of their effect estimate."

Indeed, by adopting a ±10% instead of the ±5% standard the EPA report aims at giving an artificial impression of the stability and certainty of the data. By this gambit the data are not changed, but their intrinsic uncertainty is fictitiously reduced. Experts may see through this machination, but also understand that its only possible purpose is to pull some wool over less sophisticated eyes.

In a separate and less known document the EPA states:

> "Our best estimate is about 3000 deaths a year. While the true number may be higher or lower, the totality of the evidence and especially the groups receiving the highest exposure levels strongly argue that the number is greater than zero."[7]

In fact, only by artificially increasing the stability of the data and ignoring contrary evidence could the EPA make this statement, because the "totality of the evidence" is also compatible with numbers less than zero.

My purpose here is not to cast aspersions on ETS studies, but rather is intended to illustrate how contradictory and essentially uninterpretable are the results if analyzed comprehensively in a true "weight of evidence" exercise. In fact, the "weight of evidence" of which the EPA report speaks is nothing but an exercise in selective use of data, and therefore not a "weight of evidence" analysis at all.

Only by selectively choosing data that suit a preconceived policy could one arrive at the conclusions that are incorrectly represented as scientific evidence to the public and to you, members of the subcommittee, by Stockwell et al., Brownson et al., most other authors of ETS epidemiologic studies, the U.S. Surgeon General, The National Academy of Sciences, the EPA and, of course, Dr. Munzer and other advocates.

A major justification advanced is that by now several studies have arrived at similar conclusions. However, this is true only if one eliminates all the contrary data and ignores that most studies suffer from the same defects and shortcomings. Illusory attempts have been made to increase the significance of these studies by combining their data in what is known as a meta-analysis exercise. Yet, it is intuitively difficult to improve the overall quality of conclusions by combining individual studies that are intrinsically flawed. Rotten apples could hardly make good applesauce.

In the ETS report, the EPA has no hesitation in disclosing that their analysis "is based on the a priori hypothesis ... that a positive association exists between exposure to ETS and lung cancer"[8]. On this basis, as the agency further proceeds to disregard systematically all evidence that contradicts its a priori assumption, it may as well start trying witches again.

And trying witches again it will, if Congress and our collective social culture let the agency get away with this arbitrary use of selected evidence.

There is a simple remedy: if the EPA wishes to claim that its policies are based on scientific evidence, then it should follow those rules of scientific evidence that any scientific institution or individual scientist is expected to observe.

Indeed it is strange that at a time when scientific misconduct is hotly debated in our midst and is the burning focus of Congressional hearings, official agencies of our own government are praised and feel free and proud to operate under scientific pretensions that would bring swift reprisal to any outside scientist or institution.

Mr Chairman, Members of the Subcommittee, the real issue in front of you is not passive smoking, as you may think. In reality, you are truly deciding whether our society shall in the end become hostage to less than benevolent official powers.

REFERENCES

1. Environmental Protection Agency. Respiratory health effects of passive smoking: lung cancer and other disorders. EPA, Washington, DC, 1992

2. Stockwell, HG, et al., Environmental tobacco smoke and lung cancer risk in nonsmoking women, JNCI 84:1417-1422, 1992

3. Brownson, RC, et al., Passive smoking and lung cancer. AJPH 82:1525-1530, 1992

4. See exhibit 1 (from reference 2)

5. See exhibit 2 (from reference 3)

6. See exhibit 3 (from reference 3)

7. Letter of Dr. Erich W. Bretthauer, Assistant Administrator for Research and Development, EPA, to Mr. J.J. Tozzi. December 17, 1992

8. Reference 1, page 5-2

Table 3. Effect of environmental tobacco smoke on lung cancer risk of nonsmoking women, according to source of case patient interview

Exposure history	Source of case patient interview							
	Self (n = 70)		Husband (n = 48)		Other surrogate (n = 92)		Self and husband (n = 118)	
	OR*	95% CI	OR*	95% CI	OR*	95% CI	OR*	95% CI
Exposure: yes/no								
Father	3.2	0.9-11.3	2.0	0.4-8.8	0.6	0.3-1.3	2.7	1.0-7.3
Husband	3.1	0.9-10.6	3.1	0.7-13.7	0.9	0.4-1.9	3.1	1.2-8.3
Siblings and others	—	—	—	—	—	—	4.3	1.3-14.2
Smoke-years								
Childhood/adolescence (parents and siblings)								
<18	4.3	1.1-16.7	2.4	0.4-12.8	0.6	0.2-1.9	3.6	1.2-10.8
18-21	2.4	0.5-10.6	1.6	0.2-10.5	0.6	0.2-2.0	2.1	0.6-7.2
≥22	6.5	1.7-25.5	1.4	0.2-9.6	1.6	0.5-4.8	4.4	1.4-13.5
P for trend		.04		.78		.68		.07
Adulthood (husband and others)								
<22	3.4	0.9-12.2	3.1	0.7-14.8	0.8	0.3-2.0	3.3	1.2-9.2
22-39	3.6	1.0-13.1	1.8	0.4-9.2	0.8	0.3-1.9	2.9	1.0-8.2
≥40	4.7	1.2-19.1	4.2	0.8-22.9	1.5	0.6-3.9	4.7	1.5-14.7
P for trend		.02		.05		.35		.006
All lifetime household exposure								
<22	2.0	0.5-7.6	3.2	0.7-14.9	0.6	0.3-1.6	2.5	0.9-7.2
22-39	4.4	1.3-15.6	1.0	0.2-5.6	0.6	0.2-1.4	3.1	1.1-8.6
≥40	4.1	1.1-14.8	3.5	0.7-16.7	1.5	0.7-3.6	4.0	1.4-11.3
P for trend		.01		.15		.11		.004

*OR, adjusted for age, race, and education (≤8 and >8 grades).

TABLE 1—Adjusted Odds Ratios (OR)[a] and 95% Confidence Intervals (CI) for the Relationship between Passive Smoke Exposure during Childhood and Lung Cancer in Women, Missouri, 1986 through 1991

Source of Exposure	All Subjects[b]				Lifetime Nonsmokers			
	No. Cases	No. Controls	OR	95% CI	No. Cases	No. Controls	OR	95% CI
All household members								
Never	430	928	1.0		323	802	1.0	
Ever	185	472	0.8	0.7, 1.1	108	364	0.8	0.6, 1.1
Cigarette pack-years								
0	430	928	1.0		323	802	1.0	
>0–15	42	129	0.7	0.5, 1.0	27	104	0.7	0.4, 1.1
>15–25	31	119	0.6	0.4, 0.9	20	91	0.6	0.4, 1.0
>25	34	117	0.7	0.4, 1.1	21	87	0.7	0.4, 1.2
Parents only								
Never	489	1021	1.0		357	877	1.0	
Ever	126	379	0.7	0.5, 0.9	74	289	0.7	0.5, 0.9
Cigarette pack-years								
0	489	1021	1.0		357	877	1.0	
>0–15	19	90	0.4	0.3, 0.7	12	70	0.5	0.2, 0.8
>15–25	27	118	0.5	0.3, 0.7	17	87	0.5	0.3, 0.9
>25	33	99	0.7	0.5, 1.1	21	74	0.8	0.5, 1.4

[a]Adjusted for age, history of previous lung disease, and active smoking (all subjects only).
[b]Includes lifetime nonsmokers and ex-smokers who had stopped at least 15 years before diagnosis or who had smoked for less than 1 pack-year.

TABLE 2—Adjusted Odds Ratios (OR)[a] and 95% Confidence Intervals (CI) for the Relationship between Passive Smoke Exposure during Adulthood and Lung Cancer in Women, Missouri, 1986 through 1991

Source of Exposure	All Subjects[b]				Lifetime Nonsmokers			
	No. Cases	No. Controls	OR	95% CI	No. Cases	No. Controls	OR	95% CI
All household members								
Never	221	527	1.0		170	470	1.0	
Ever	394	873	1.0	0.8, 1.2	261	696	1.1	0.8, 1.3
Cigarette pack-years								
0	221	527	1.0		170	470	1.0	
>0–15	88	234	0.9	0.6, 1.2	56	181	0.9	0.6, 1.2
>15–40	91	261	0.8	0.6, 1.0	62	199	0.9	0.6, 1.2
>40	146	264	1.3	1.0, 1.6	107	217	1.3	1.0, 1.8
Cigarette pack-years × hours/day[c]								
0	221	527	1.0		170	470	1.0	
>0–50	90	261	0.8	0.6, 1.1	63	206	0.9	0.6, 1.2
>50–175	89	246	0.8	0.6, 1.1	58	189	0.9	0.6, 1.2
>175	124	238	1.2	0.9, 1.6	92	192	1.3	1.0, 1.8
Spouse only								
Never	287	650	1.0		213	568	1.0	
Ever	328	750	0.9	0.8, 1.1	218	598	1.0	0.8, 1.2
Cigarette pack-years								
0	287	650	1.0		213	568		
>0–15	58	166	0.7	0.5, 1.0	32	128	0.7	0.5, 1.1
>15–40	81	258	0.7	0.5, 0.9	54	200	0.7	0.5, 1.0
>40	150	266	1.2	0.9, 1.5	110	216	1.3	1.0, 1.7
Cigarette pack-years × hours/day[c]								
0	287	650	1.0		213	568	1.0	
>0–50	64	201	0.7	0.5, 0.9	41	161	0.7	0.5, 1.0
>50–175	81	237	0.7	0.5, 1.0	52	183	0.8	0.5, 1.1
>175	126	241	1.1	0.9, 1.5	94	193	1.3	1.0, 1.7

[a]Adjusted for age, history of previous lung disease, and active smoking (all subjects only).
[b]Includes lifetime nonsmokers and ex-smokers who had stopped at least 15 years before diagnosis or who had smoked for less than 1 pack-year.
[c]The product of total pack-years and average number of hours exposed per day to passive smoke in the home.

HARVARD SCHOOL OF PUBLIC HEALTH
Department of Environmental Health
Environmental Epidemiology Program

Prepared testimony by Douglas W. Dockery, Associate Professor of Environmental Epidemiology, April 22, 1993.

Hearing on H.R. 881, a bill to ban smoking in all Federal Buildings by the Committee on Public Works and Transportation, Subcommittee on Public Buildings and Grounds.

Tobacco smoke is a known cause of lung cancer. If tobacco smoke were held to the same standards applied to other environmental carcinogens found in public buildings, there is no question it would be banned. We consider it unacceptable to passively expose people in public buildings to asbestos, formaldehyde, or radon. It therefore should not be acceptable to passively expose people in public buildings to tobacco smoke.

The carcinogeniticity of tobacco smoke has been demonstrated by all methods used to assess risk, that is in animal bioassay studies, genotoxicity studies, and epidemiologic studies. Tobacco smoke is not only a recognized cause of cancer, it is a strong carcinogen. Epidemiologic studies have shown that active smokers develop lung cancer at a rate at least ten times that of never smokers. Epidemiologic studies have shown that the risk of lung cancer associated with tobacco smoke increases with exposure, either as measured by number of cigarettes smoked per day, or years of cigarette smoking. There is no evidence from these studies of active smokers that even the smallest exposures to active smoking are free of risk. It follows immediately that exposures to low concentrations of tobacco smoke should be associated with increased risk of lung cancer. **The evidence that tobacco smoke is such a strong cause of lung cancer, without any other consideration, is sufficient to define environmental tobacco smoke as a lung cancer hazard and to ban smoking from all Federal Buildings.**

There is also substantial evidence showing that nonsmokers are passively exposed to tobacco smoke at nontrivial levels. A considerable mass of data has been developed estimating environmental exposures to tobacco smoke, direct measurements of ambient indoor concentrations of tobacco smoke, and direct measures of dose based on biologic tissue samples. Non-smokers have levels of cotinine, a specific nicotine metabolite, which are 1/10% to 2% of those of smokers. **The evidence that large numbers of people in the general population are exposed to this carcinogen is sufficient to define environmental tobacco smoke as a lung cancer hazard and to ban smoking from all Federal Buildings.**

Given that tobacco smoke is associated with increased incidence of lung cancer down to the lowest exposures among active smokers, and that there is widespread environmental exposure to tobacco smoke among nonsmokers, increased lung cancer incidence should be expected among never smokers chronically exposed to environmental tobacco smoke. Indeed, increased incidence of lung cancer has been consistently observed in studies of never smoking women married to husbands who smoke. **The evidence from epidemiologic studies that never smoking women with smoking spouses have increased risk of lung cancer only confirms what was apparent from the carcinogenicity of tobacco smoke itself.**

Given the compelling evidence cited above, the hypothesis to be tested (that is, the null hypothesis) is not whether environmental tobacco smoke has a statistically significant association with lung cancer in these epidemiologic studies. Our prior hypothesis would certainly be that environmental tobacco smoke increases the risk of lung cancer. Thus the hypothesis to be tested is that environmental tobacco smoke does not increase lung cancer risk. Of the 30 studies reviewed by the EPA[1], only one study[2] was not consistent with an increased lung cancer risk associated with spousel smoking. The consistency of these results across so many independent studies showing a positive association between lung cancer and

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[1]Environmental Protection Agency, "Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders." EPA/600/6-90/006B, May 1992.

[2]Wu-Williams AH, Samet JH. Environmental tobacco smoke: exposure response relationships in epidemiologic studies. Risk Analysis. 1990:10:1.

spousal smoking is a very strong statement of the robustness of those findings. In epidemiologic studies of effects of environmental hazards such consistency and robustness is a much more important indicator of causality than statistical significance.

The attached figure presents my summary of the available epidemiology data showing the association of lung cancer in non-smoking women with cigarette smoking of their husbands. Results from 32 studies are presented. For each study, the estimated relative odds of lung cancer associated with husband's cigarette smoking is presented along with the 95% Confidence Interval for that estimate. A relative odds greater than one, that is on the right side of the plot, indicates that lung cancer in these nonsmoking women is positively associated with their husbands' smoking. It is clear from this plot that almost all of these studies have found such a positive association.

Critics have suggested that these associations may be the result of bias or confounding in the data. That is, the observed associations are due to some characteristic other than spousal smoking which is related to <u>both</u> lung cancer and spousal smoking. While each study has weaknesses, and bias and confounding must be considered in any study, it is my interpretation of these studies that bias and confounding have generally acted to underestimate the true association between lung cancer and environmental tobacco smoke exposure. Other design issues, such as the fact that spousal smoking is only a crude measure of environmental tobacco smoke exposure in the home, at work, and in other settings, will also produce underestimates of the true association.

Critics have also suggested that the use of meta-analytic techniques, as in the EPA Review, are inappropriate for epidemiologic data. In this I strongly disagree. Meta-analysis is a well developed method for contrasting and combining results from different studies. The concepts and methods are actually a simple extension of statistical analysis commonly used in epidemiology. These methods are commonly used in public health research, particularly

in the evaluation of clinical trials[3]. Meta-analysis has been applied in epidemiologic research for many decades[4] and has recently been reviewed in two scholarly reviews[5,6]. The use of these methods by the EPA is not only appropriate, but is a significant advance in evaluating the epidemiologic literature.

In my opinion, the epidemiologic evidence for an association between lung cancer and environmental tobacco smoke is very compelling. The EPA Review is a comprehensive, rigorous, balanced and scholarly summation of the current state of the science, which supports such a finding. While epidemiologic studies alone cannot demonstrate causality, the universal finding of the carcinogenicity of tobacco smoke in animal and genotoxicity studies corroborates the epidemiology. There is no doubt that tobacco smoke is an environmental carcinogen, which should be banned from Federal Buildings and all other public facilities.

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[3]Louis TA, Fineberg HV, Mosteller F. Findings for public health from meta-analysis. Annual Reviews of Public Health 1985; 6:1-20.

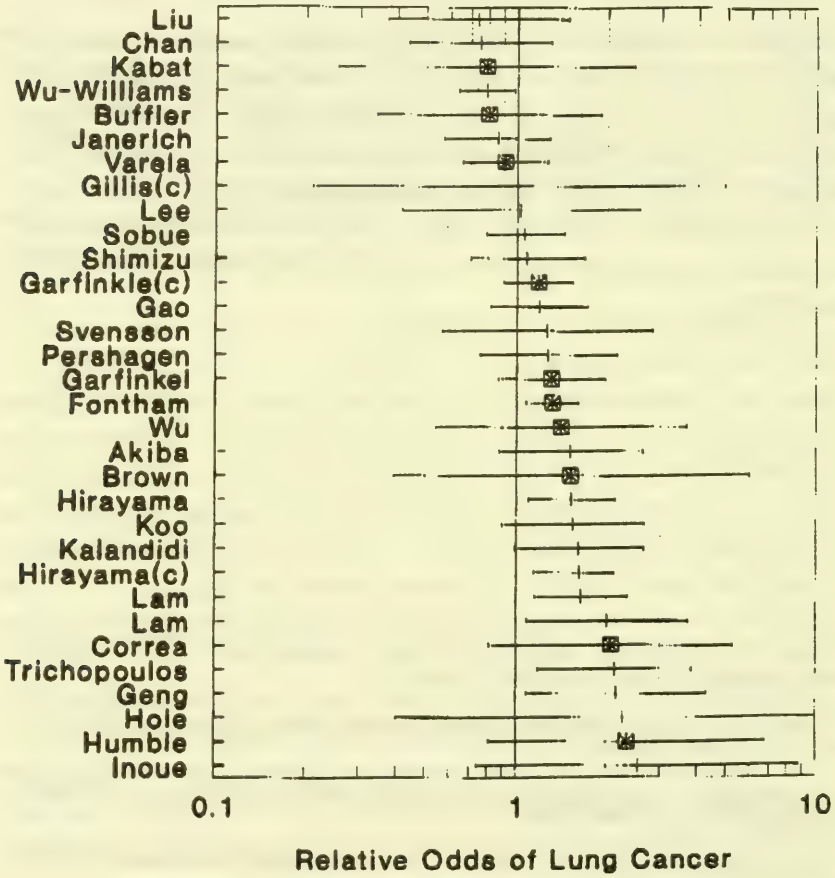
[4]MacMahon B, Hutchinson GB. Prenatal x-ray and childhood cancer: a review. Acta Un Int Cancer 1964; 2.1172-1174.

[5]Greenland S. Quantitative methods in the review of epidemiologic literature. Epidemiologic Reviews 1987; 9:1-30.

[6]Dickersin K, Berlin JA. Meta-analysis: State-of-the-Science. Epidemiologic Reviews 1992; 14:154-176.

EPIDEMIOLOGIC STUDIES OF PASSIVE SMOKING & LUNG CANCER

Studies



BIOSKETCH

Dr. Douglas W. Dockery is Associate Professor of Environmental Epidemiology at the Harvard School of Public Health and Assistant Professor of Medicine (Epidemiology), Channing Laboratory, Harvard Medical School and Brigham and Women's Hospital. He was born in Washington, D.C. *(on Capitol Hill)* and received his BS in Physics from the University of Maryland at College Park. He has a MS in Meteorology from MIT and a ScD (Doctor of Science) in Environmental Health from the Harvard School of Public Health. Dr. Dockery is conducting epidemiologic research on the natural history of respiratory disease and the respiratory health effects of air pollution. He is principal investigator of the Six Cities study of the longitudinal health effects of sulfur oxide and particulate exposures among adults and children in the United States, and co-principal investigator in the Twenty-Four Cities study of the effects of acid aerosols on respiratory health of children in the United States and Canada. In addition, he is collaborator or advisor in studies of the health effects of air pollution in the Netherlands, Switzerland, Germany (East and West), Czechoslovakia, Brazil, Mexico City, and Beijing. Dr. Dockery has more than 45 published articles, primarily describing epidemiologic studies of the health effects of air pollution, both outdoors and indoors, on children and adults. In addition, Dr. Dockery and his colleagues have published on the normal growth and decline of pulmonary function, the effects of smoking (active and passive) on respiratory health, and measurement of exposure for environmental epidemiologic studies. Dr. Dockery is also working on synthesizing data from epidemiologic studies of environmental health effects from independent studies using meta-analytic methods. He teaches graduate courses on *Respiratory Epidemiology* and *Environmental and Occupational Epidemiology* at the Harvard School of Public Health.

Society of Critical Care Medicine

TESTIMONY OF JOHN H. HOYT, MD, FCCM ON BEHALF OF THE SOCIETY OF CRITICAL CARE MEDICINE
BEFORE THE HOUSE PUBLIC WORKS AND TRANSPORTATION COMMITTEE
SUBCOMMITTEE ON PUBLIC BUILDINGS AND GROUNDS
TESTIMONY IN SUPPORT OF H.R. 881,
BANNING SMOKING IN FEDERAL AND CONGRESSIONAL BUILDINGS

April 22, 1993

Mr. Chairman and Members of the Subcommittee, I am John Hoyt, MD, FCCM, Chairman of the Critical Care Medicine Department and Medical Director of the Medical/Surgical Intensive Care Unit (ICU) at St. Francis General Hospital in Pittsburgh, Pennsylvania. St. Francis General Hospital is a 750-bed, full-service facility that has provided care to patients in Pittsburgh for 125 years. I am also a clinical professor of anesthesiology and critical care at the University of Pittsburgh, part of the largest fellowship training program of critical care physicians in the world.

I am a board-certified critical care medicine physician. My medical practice focuses on the care of individuals who suffer from immediate life-threatening illness and injury. My patients develop lung, brain and heart failure that is usually a result of trauma, extensive surgery, overwhelming infection, heart attack, stroke and poisoning that occurs from tobacco smoke.

My testimony today is presented as Treasurer of the Society of Critical Care Medicine, an organization comprised of more than 7,500 physicians, nurses and allied health care practitioners who are committed to the care of the approximately 4 to 6 million Americans who become critically ill or injured each year. Members of the Society are, unfortunately, all too well aware of the significant health care consequences of prolonged smoking or exposure to second-hand smoke that cost this country significant health care resources each year. Smoking kills nearly 400,000 Americans annually, including 115,000 from coronary artery disease; in general, smokers double their risk of heart disease compared to non-smokers; while those smokers between the ages of 45 and 64 years of age are three times more likely than non-smokers to suffer from heart disease. Another 27,000 Americans die from stroke, 136,000 from cancer and 50,000 from other related illnesses. A survey published by the Society of Critical Care Medicine reported that on any given day, 14.6 percent of all ICU admissions are diagnosed with respiratory insufficiency or failure. Every year, $22 billion are spent on the medical costs related to illnesses caused by the addiction to tobacco. Medicare and Medicaid spend $4.2 billion annually for the care of patients with tobacco-related illnesses.

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Recently, I experienced one of the busiest nights in the ICU I can recall in my 17-year career as a critical care intensivist. Of the eight patients I admitted to the ICU that night, six were admitted with smoking-related illnesses and needed the support of a ventilator to breathe. Their disease, Chronic Obstructive Pulmonary Disease (COPD), literally obstructs the workings of the lung. The lung consists of a delicate architecture of tubes and sacs, that allow gas exchange and oxygen delivery. Smoking leads to damage of these tubes and sacs and obstructs the flow of oxygen to the lung. COPD causes inadequate function of the lung, shortness of breath, limited exercise ability, the need for support from mechanical ventilation and finally death from respiratory failure. The process is long and debilitating; many COPD patients suffer for as long as five years before dying of this disease. Their care is expensive; the cost for a patient to receive support from a ventilator is eight times more than average hospital care. According to the Centers for Disease Control, sixty thousand Americans die each year of Chronic Obstructive Pulmonary Disease.

How do tobacco-related costs add up in the ICU? My colleague, Dr. Timothy Buchman, Co-Director of the Surgical ICU at Johns Hopkins Hospital in Baltimore, recently published a study in the journal *Chest*, of admissions to his unit which showed that tobacco, alcohol and illegal drug-related illnesses and injuries accounted for 28 percent of the admissions and 29 percent of the medical costs during a 15-week period. The 435 patients admitted to this particular ICU generated $3,014,953 in costs in a unit with a per night cost of $3,000 per bed. The 59 patients with tobacco-related illnesses alone accounted for $481,648 in costs, or approximately $8,000 each. Tobacco-related cases that involved cancer and respiratory diseases accounted for 14 percent of the admissions and 16 percent of the costs. All but one of the 59 cases were related to cigarette smoking; one case was related to cigar smoking.

The physical and societal ills associated with smoking affect not only adults, but our children as well. A 1991 report from the Department of Health and Human Services reported that half of all eighth graders and nearly two-thirds of all tenth graders have tried cigarettes. Infants are also adversely affected; 20 to 30 percent of low birth weight babies, up to 14 percent of preterm deliveries and approximately 10 percent of all infant deaths are attributable to cigarette smoking during pregnancy.

The life-threatening effect of passive smoking has been well documented. The recent EPA report, "Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders" clearly stated the dangers of breathing second-hand smoke. When inhaled, environmental tobacco smoke is a human carcinogen that can be directly blamed for the lung cancer deaths of 3,000 non-smokers each year in the U.S. In children, second-hand smoke clearly increases the risk of lower respiratory tract infections, including bronchitis and pneumonia, resulting in the hospitalization of 7,500 to 15,000 infants and children each year. This carcinogen also increases the prevalence of middle ear disease and the frequency and

severity of symptoms in asthmatic children. According to the EPA, the condition of 200,000 to 1 million asthmatic children is worsened by exposure to second-hand smoke. The Society of Critical Care Medicine agrees with former EPA Administrator William K. Reilly's statement that environmental tobacco smoke may present the most important environmental health risk facing Americans today.

In this era of historic health care reform, where quality of care, access to care and cost of care are such intensely debated issues, these results - - which are due to ***preventable*** illness - - are entirely unacceptable. The "common courtesy" approach to eliminating passive smoking that is advocated by the tobacco industry has not worked. Policymakers, health care providers and patient advocates, all must ensure that the health of Americans is not further endangered by tobacco smoke. We must ensure that scarce and expensive health care resources are allocated in the most efficient manner possible. Too many other *unpredictable and unpreventable* illnesses and injuries require our attention, including overwhelming infection, traumatic injury, multi organ system failure and others. Improving the health of all Americans, by eliminating smoking and the diseases it causes, will prolong life and release the precious human, physical and financial resources needed to care for other patients.

The Society of Critical Care Medicine supports H.R. 881, which would establish a complete ban on smoking in all federal and Congressional buildings. Serious health problems caused by smoking have been well documented over a number of years. We believe it is time for Congress to take a stand and take the lead on this important issue by banning smoking in all private offices and public areas of federal and Congressional buildings.

Mr. Chairman, members of the Subcommittee, thank you for the opportunity to present the Society's views on this very important issue. We look forward to working with Congress and Members of the Subcommittee to eliminate this life-endangering threat to the lives of all Americans, adults and children. I will be happy to answer any questions you or the Subcommittee Members may have at this time.

American Heart Association

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Coalition on Smoking OR Health

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Association of State and Territorial Health Officials
March of Dimes Birth Defects Foundation

TESTIMONY OF ALFRED MUNZER, M.D.

ON BEHALF OF

THE COALITION ON SMOKING OR HEALTH

to the

House Committee on Public Works and Transportation

Subcommittee on Public Buildings and Grounds

U.S. House of Representatives

RE: H.R. 881, LEGISLATION TO BAN SMOKING IN FEDERALLY OWNED AND LEASED OFFICE BUILDINGS

April 22, 1993

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Mr. Chairman, and members of the subcommittee on Public Buildings and Grounds, I am Dr. Alfred Munzer, President-elect of the American Lung Association (ALA). I am also Director of Critical Care and Pulmonary Medicine at Washington Adventist Hospital in Takoma Park, MD, where I specialize in the treatment of diseases of the lung.

The ALA is the nation's oldest voluntary health organization and is dedicated to the prevention and control of lung disease. This organization, and its medical section, the American Thoracic Society, has long recognized the contribution of indoor and outdoor air pollution to the development and exacerbation of lung disease. The ALA has devoted the past 26 years to the implementation of programs aimed at improving air quality in our homes and in our communities.

Today I am testifying of behalf of the American Lung Association, the American Cancer Society, and the American Heart Association, united as the Coalition on Smoking OR Health. Formed in 1982, the coalition has worked to heighten public awareness about the impact of tobacco consumption upon public health. It believes strong measures should be imposed to discourage tobacco use in all segments of the population, including youth, women, and minorities.

As a pulmonary physician, I all too often see first hand the devastation caused by tobacco use. I see the men and women who come to me with end-stage lung cancer or emphysema, seeking a medical miracle to cure their disease. I see the children who cough and wheeze as their asthma is made worse by exposure to smoke exhaled by smokers and that comes from the burning end of a cigarette, pipe, or cigar. Smoke of this nature is commonly called involuntary, passive, or secondhand smoke. However, more recently, it is increasingly referred to as "ETS", or environmental tobacco smoke.

I cannot express to you how critical it is for us to respond to the ETS issue. Conclusions drawn from the Environmental Protection Agency's (EPA) risk assessment on ETS reenforces the sense of urgency in this regard. If we do not take immediate action, or ignore its impact on public health, ETS can easily be the cause of approximately 3,000 lung cancer deaths in nonsmokers in the coming year. I am certain this is not the future trend our society desires to establish.

Today, I do not intend to argue whether smokers should have a right to smoke it public, nor am I here to urge the subcommittee to revoke this privilege. However, I have elected to appear before you due to ongoing concern regarding the health effects of ETS for nonsmokers and particularly children, and the need to impose stringent measures, both in

government and the private sector, to adequately address this growing public health concern.

I want to begin by reflecting on the evidence which has supported and recently led to the EPA findings on ETS, and move into a examination of the methodology which supports this agency's assertion that ETS is a carcinogen. I will speak briefly to the claims raised by tobacco advocates regarding the validity of the EPA findings, and lastly, focus on public interest in and support for significant action to limit or eliminate exposure to ETS is public areas.

ETS has been the topic of discussion for more than 20 years. Its health effects were first reviewed in 1972 in the U.S. Surgeon General's report on smoking and health. That report was devoted, in part, to public exposure to air pollution caused by tobacco smoke. It concluded that "an atmosphere contaminated with tobacco smoke can contribute to the discomfort of many individuals."

In 1982, the U.S. Surgeon General <u>again</u> examined the issue of passive smoking, but this time in the context of smoking and the development of cancer. At that time there were only 3 epidemiologic studies linking passive smoking and lung cancer. Even with this limited amount of evidence, the Surgeon General concluded that the evidence in these studies

is the cause for grave concern regarding the possible serious public health problem associated with passive smoke and lung cancer.

By 1986, federal interest in the health effects of ETS had grown to the extent that the U.S. Surgeon General released a report devoted entirely to the issue of passive smoking. By that time, the number of epidemiologic studies had increased to 13, 11 of which showed a positive correlation between passive smoking and lung cancer in healthy nonsmokers. Based upon these findings, the Surgeon General concluded that exposure to secondhand smoke is a cause of lung cancer in healthy nonsmokers. He also concluded that children whose parents smoked had an increased frequency of respiratory symptoms and infections, compared to children whose parents were nonsmokers.

Several private organizations -- the National Academy of Science and the International Agency for Cancer Research -- published reports which drew conclusions similar to those of the EPA. The International Agency for Cancer Research, for example, released a report on cancer which concluded that "knowledge of the nature of sidestream and mainstream smoke, of materials absorbed during passive smoking, and of the quantitative relationships between dose and effect that are commonly observed from exposure to carcinogens leads to the

conclusion that passive smoking gives rise to some risk of cancer."

Shortly after the release of these studies, the EPA began to examine the health effects of passive smoking on children and adults. The agency issued an initial analysis of the risks of exposure to ETS in May, 1990. Entitled, "Health Effects of Passive Smoking: Assessment of Lung Cancer in Adults and Respiratory Disorders in Children," the risk assessment focused on the potential correlation between ETS and lung cancer in nonsmoking adults and respiratory disease and pulmonary effects in children.

On January 7, 1993, the EPA released its final report assessing current scientific evidence on the risks of exposure to ETS. Based on the total weight of evidence in the scientific literature, the EPA designated ETS as a Group A carcinogen, a rating used only for extremely hazardous substances known to cause cancer in humans. It ranked ETS in a class of carcinogens which includes asbestos, benzene, and radon.

After evaluating 30 epidemiological studies on lung cancer in nonsmoking adults, the EPA determined that ETS is responsible for approximately 3,000 lung cancer deaths each year. The agency also added that ETS accounts for the development of 20

percent of all lung cancers caused by factors other than smoking. For the average adult, ETS increases their risk of cancer to approximately 2 per 1,000. From these conclusions, it is clear that ETS is far more hazardous to the health of nonsmoking adults.

After evaluating more than 100 studies on respiratory health in children, the EPA concluded that ETS exposure increases their risk of lower respiratory infections, like bronchitis and pneumonia. ETS is known to cause an estimated 150,000 to 300,000 cases of respiratory illnesses in children up to 18 months each year. Of these cases, 7,500 to 15,000 result in hospitalization.

ETS exposure is also associated with additional attacks and increased severity of symptoms in children with asthma. The EPA estimates that 200,000 to 1 million asthmatic children have their condition worsened by ETS, and that ETS is a risk factor for new cases of asthma in children without a history of symptoms.

Also of concern are the risks for children whose mothers smoked during and after pregnancy. The U.S. Department of Health and Human Services has reported that, under these circumstances, children are three times more likely to die of Sudden Infant Death Syndrome (SIDS) than children of

nonsmoking mothers. The risks of SIDS double for children whose mothers smoked after birth and not during pregnancy than for children reared in nonsmoking environments.

The evidence presented represents very sound science and more than adequately supports the conclusions by the EPA regarding exposure to ETS. Uniquely, each of the studies and reports used to reach this conclusion were developed and edited by different processes. In contrast to assertions made of opponents of the EPA's findings, such as those offered by the tobacco industry, it is this diverse methodology which only strengthens the validity of the conclusion of this research combined.

Without spending too much time on the tobacco industry's criticisms of the risk assessment, let me first remind the subcommittee that after 60,000 studies linking smoking with disease and death, this industry still fails to acknowledge that it produces a lethal product. This is an industry which has criticized each Surgeon General's report since 1964. Among the industry criticisms is the failure of the EPA to include studies which show no relationship between ETS and lung cancer. Among the studies cited by the industry as examples are several funded by the National Cancer Institute:

- Brownson, PhD., et.al. Passive Smoking and Lung

Cancer in Nonsmoking Women. Am J Public Health 82:1525-1530, 1992.

This study was published in November 1992, too late for inclusion in the risk assessment unless it was further delayed. The industry contends that the risk assessment would change if the study were included. However, the author's of the study conclude: "Ours and other recent studies suggest a small but consistent increase risk of lung cancer from passive smoking. Comprehensive actions to limit smoking in public places and worksites are well-advised."

♦ Stockwell, Sc.D., et.al. Environmental Tobacco Smoke and Lung Cancer in Nonsmoking Women. J Natl Cancer Inst 84:1417-1422, 1992.

This study was not included in the final risk assessment and again the industry claims it is a negative study therefore left out purposefully. However, the author's conclude: "These findings suggest that long-term exposure to environmental tobacco smoke increases the risk of lung cancer in women who have never smoked."

The real issue here is statistical significance and how it is used. In defining the true meaning of statistical

significance, I'd like to defer to the description used by a well-noted environmental epidemiologist, Dr. Douglas Dockery, and Associate Professor at the Harvard School of Public Health. Dr. Dockery suggests:

> " A naive critique would say that those studies which are not 'statistically significant' do not show an effect. However, statistical significance is not a measure of association or environmental tobacco smoke with lung cancer, but rather a measure of the stability of the association. It measures the statistical power of the study. In a crude sense it is a measure of study size, and studies that do not achieve statistical significance are simply too small. This does not mean that they do not provide important information on risks. It is not appropriate to discard studies which do not achieve statistical significance, but rather they should be included giving them a weight which reflects the stability, that is the uncertainty, of their effect estimate. This is exactly what the meta-analysis of these studies provides."

Mr. Chairman, we at the Coalition on Smoking OR Health believe the EPA's findings are clear, objective, and complete in regard to ETS. The evidence used to show the relative risks associated with exposure to ETS, and its linkage to the development of lung cancer, are more compelling than similar

correlations drawn for other environmental carcinogens. I hope the evidence I have presented to the subcommittee today will enable you to step beyond the criticisms offered regarding the validity of the EPA risk assessment, and encourage to you move forward in your efforts to address the real issue on the table -- adequately responding to the public health issue associated with exposure to ETS, particularly in those places where people spend a lot of time.

Let me commend the subcommittee for the effort and commitment it has made to this issue, thus far. The introduction and careful review of legislation to ban smoking in federal buildings is a very important step in this process. The Coalition on Smoking OR Health supports the bill -- H.R. 881 -- on which this hearing is being held today, and similar proposals which have been offered by other Members of Congress this year.

In examining the proposal to ban smoking in federal buildings, let me remind you that the federal government has taken little initiative to protect federal workers from exposure to secondhand smoke. Any action taken to date remains inconsistent with each federal agency responsible for its own policy. The General Services Administration (GSA) is reconsidering its regulations, but GSA space accounts for

only 10 percent of federal building space.

I urge this subcommittee to take into consideration growing interest in smoke-free public places, which has gained momentum since the release of the EPA risk assessment. Based on a public opinion survey conducted by the American Lung Association, it is clear that more and more Americans believe ETS is harmful, and that they prefer smoke-free public places, as opposed to those environments in which smoke is permitted.

In this survey, the Lung Association found that 8 in 10 smokers know that ETS is bad for the people around them. We also found that nonsmokers are more likely than smokers to strongly agree about the harmful effects of ETS exposure. The survey found increasing support for total bans on smoking in public places such as restaurants, workplaces, hotels, buses, and trains. And though current smokers are more likely than others to believe that smoking in public places should be restricted, very few smokers surveyed favored no restrictions.

Clearly, as the awareness of the health hazards of ETS increases, more Americans are striving to live, work, and breathe in smoke-free environments. Very few of us make it through each day without exposure to ETS. Those who are

confined to indoor environments -- like employees and staffers in the House of Representatives -- are no exception. It is unfortunate that the House Building Commission's recent decision to merely restrict smoking to certain areas does not provide the adequate protection needed.

I believe the 1986 report of the Surgeon General has the best recommendation for us to consider. In its conclusion, the report clearly states, "Simple separation of smokers and nonsmokers within the same air space may reduce, but does not eliminate, exposure of nonsmokers to ETS." Therefore, it is the responsibility of employers and employees to "ensure that the act of smoking does not expose the nonsmoker to tobacco smoke" and for smokers to "assure that their behavior does not jeopardize the health of other workers." In addition, the Surgeon General stated that nonsmokers have the "responsibility to provide a supportive environment for smokers who are attempting to stop."

Congress owes its employees and the people of this country who frequent the Capitol grounds to provide a healthy and safe environment. By going smoke-free, the House of Representatives and the Senate will contribute to the notion that nonsmoking is a social norm.

On behalf of the American Lung Association, and other members

of the Coalition on Smoking OR Health, I would like to thank you for the opportunity to testify before the subcommittee on the impact of ETS exposure upon public health. Again, we urge you to step beyond the criticism offered regarding the validity of the EPA data and take into account the points we have raised today, which demonstrate a need for government action. Please know you have our organization's support and encouragement as you continue to review this very pressing issue.

AHA Medical/Scientific Statement

Position Statement

Environmental Tobacco Smoke and Cardiovascular Disease

A Position Paper From the Council on Cardiopulmonary and Critical Care, American Heart Association

Aubrey E. Taylor, PhD, Chairman; Douglas C. Johnson, MD,
and Homayoun Kazemi, MD, Members

Cigarette smoking was identified by the Surgeon General in 1982 and 1983 as the most important modifiable risk factor for cancer and chronic heart disease in the United States.[1,2] Recent studies have implicated exposure to environmental tobacco smoke as a significant risk factor for the development of lung cancer and heart disease. Because more information on environmental tobacco smoke is now available, its health effects are reviewed in this report, with a major emphasis on the relation of environmental tobacco smoke to cardiovascular disease.

Cigarette smoking has a significant effect on the health of Americans, and is a major cause of cardiovascular disease.[3] Cardiovascular disease attributable to voluntary cigarette smoking accounts for about as many deaths each year as chronic obstructive pulmonary disease and lung cancer deaths combined. In 1988 approximately 430,000 deaths in adults aged 35 and older were attributed to the intentional inhalation of tobacco smoke. This number included 201,000 deaths due to cardiovascular disease, 112,000 due to lung cancers, 83,000 due to chronic lung disease (including pneumonia, influenza, bronchitis, emphysema, chronic airway obstruction, and other respiratory diseases), and 31,000 due to other cancers.[4] It has also been estimated that an additional 3,800 lung cancer deaths[4] and 37,000 cardiovascular deaths occurred in nonsmokers who had been exposed to environmental tobacco smoke.[5] An additional 2,500 perinatal deaths were estimated to have occurred because of maternal smoking, and about 1,300 deaths resulted from burns related to smoking.[4]

Although the existing epidemiological studies on cancer deaths associated with environmental tobacco smoke may be subject to questions about sample size, exposure, experimental design, and differing lifestyles of populations, sufficient information has been published to implicate environmental tobacco smoke as a definite health hazard. The 1986 Surgeon General's report concluded that involuntary smoking is a cause of disease, including lung cancer, in healthy nonsmokers, and it was postulated that approximately 3,000–4,000 nonsmokers exposed to environmental tobacco smoke die of lung cancer each year.[6] The report also concluded that children whose parents smoke have an increased frequency of respiratory infections, increased symptoms of respiratory problems, and slightly smaller rates of increase in lung function as the lung matures compared with children of nonsmoking parents. At the time of the report, environmental tobacco smoke could not be definitely linked to cardiovascular disease. However, since 1986 several studies have been published documenting a link between environmental tobacco smoke, cancer,[7] and heart disease.[5,8] The Environmental Protection Agency has also done an extensive study of the effects of environmental tobacco smoke on lung cancer.

Environmental Tobacco Smoke

Burning cigarettes emit two types of smoke: mainstream smoke, which is the smoke directly inhaled into the smoker's lungs, and sidestream smoke, which is the smoke emitted into the air from the burning cigarette between puffs. Environmental tobacco smoke is about 85% sidestream and 15% exhaled mainstream smoke. More than 4,000 chemicals, including at least 40 carcinogens, are contained in environmental tobacco smoke.[9] Many toxic constituents are found in higher concentrations in sidestream than in mainstream smoke.[3] For example, in sidestream smoke there is about five times as much carbon monoxide (which decreases the ability of hemoglobin to carry oxygen to the tissues), three times as much benzopyrene (a tumor- and plaque-producing compound), and 50 times as much ammonia (an eye and respiratory irritant) as is inhaled directly from a cigarette. The difference is because the cigarette burns at a higher temperature during inhalation, leading to more complete combustion, and filters also screen some of these toxic compounds.

Those in close proximity to someone smoking a cigarette are exposed to smoke not only while the cigarette is lit but continue to inhale smoke that has mixed with air long after the cigarette is extinguished. Environmental tobacco smoke can persist in indoor environments for many hours after cessation of smoking, the time depending on ventilation and the mixing of

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room air with uncontaminated air.[10] To conserve energy, building ventilation rates are sometimes decreased, causing levels of smoke to increase in workplace environments, and in many homes ventilation of smoke to the outside is minimal.

Risk to Nonsmokers from Environmental Tobacco Smoke

The relative risk of developing lung cancer has been estimated to be 1.3 for nonsmokers exposed to environmental tobacco smoke at home compared with nonsmokers with no exposure to environmental tobacco smoke.[7,10,12] Active smoking has a relative risk factor for cancer of about 10.[1] Average workplace exposures to environmental tobacco smoke are estimated to increase lung cancer risk twofold because environmental tobacco smoke exposures are generally higher at the workplace than at home.[13] Despite the difficulty of interpreting epidemiological studies of exposure levels in the home and workplace, several recent studies demonstrate a definite link between cardiovascular deaths in nonsmokers exposed to environmental tobacco smoke. Glantz and Parmley[5] reviewed 10 of these studies, showing that men and women nonsmokers exposed to environmental tobacco smoke at home had an overall cardiovascular relative risk factor of 1.3. This compares to a relative risk factor of 1.7 for smokers compared with nonsmokers.[2] Kawachi et al[13] predicted an even higher relative risk factor for workplace exposures of nonsmokers to environmental tobacco smoke.

Repace and Lowrey[6] evaluated eight studies in which the number of lung cancer deaths of nonsmokers exposed to environmental tobacco smoke averaged 5,000±2,400 (mean±standard deviation) per year. Assuming that the ratio of lung cancer to heart disease deaths is the same with environmental tobacco smoke exposure as for voluntary smoking, approximately 10,000 deaths of nonsmokers exposed to environmental tobacco smoke would be expected to occur per year. However, this simple estimate does not include many aspects of environmental tobacco smoke exposure, such as the amount of environmental tobacco smoke exposure in the workplace and home, the number of persons exposed to environmental tobacco smoke, and the type and amount of smoke exposure. In fact, studies to evaluate these factors indicate that environmental tobacco smoke causes a higher risk of heart disease than predicted by this simple estimate.

Recently, Steenland[8] performed extensive analyses of the available literature on the cardiovascular effects of environmental tobacco smoke and predicted that ischemic heart disease could cause as many as 15,000–19,000 deaths yearly of nonsmokers due solely to environmental tobacco smoke from their spouses. Steenland also predicted an overall number of deaths due to environmental tobacco smoke–related cardiovascular disease of 35,000–40,000 yearly, a number similar to the number of deaths estimated by Glantz and Parmley[5] and Wells.[14] Because the risk of coronary artery disease increases markedly with the number of risk factors,[1,3,15] nonsmokers with hypertension or hypercholesterolemia and exposed to environmental tobacco smoke are likely to be at even greater risk of developing cardiovascular disease. It is well known that the risk of coronary heart disease caused by voluntary smoking decreases by about half after 1 year of smoking cessation and after several years approaches that of people who have never smoked.[16] Similar health benefits should occur in previously environmental tobacco smoke-exposed nonsmoking individuals when environmental tobacco smoke is removed from the environment in which they work and live.[9]

Exposure to Environmental Tobacco Smoke

Although the proportion of smokers in the United States is decreasing, 32% of men and 27% of women aged 20 and older smoke cigarettes.[17] These smokers will expose a vast number of nonsmokers to environmental tobacco smoke, and it has been estimated that approximately 50 million nonsmoking adults over age 35 are regularly exposed to environmental tobacco smoke.[17] Additionally, we estimate that 50% of all children live in families with one or more smokers. In a survey conducted in 1979–1980, 63% of nonsmokers reported being exposed to environmental tobacco smoke for more than 1 hour per week, 35% were exposed to environmental tobacco smoke for more than 10 hours per week, and 16% were exposed to environmental tobacco smoke for at least 40 hours per week.[18] It is likely that exposure of nonsmokers to environmental tobacco smoke has decreased in recent years because of the increased public awareness of the hazards of environmental tobacco smoke, increased restrictions on smoking areas, and better ventilation of the workplace. The public has now begun to understand the detrimental health effects of environmental tobacco smoke exposure, but this increased awareness has not eliminated exposure to environmental tobacco smoke of spouses and children living in a smoker's home or that occurring in some workplaces and public buildings.

Cardiovascular Effects of Environmental Tobacco Smoke

Environmental tobacco smoke produces acute effects on cardiovascular function in human studies. In subjects with stable angina, environmental tobacco smoke increases resting heart rate, blood pressure, and blood carboxyhemoglobin, and reduces the duration of exercise that induces angina.[19,20] Environmental tobacco smoke also produces adverse effects on the exercise performance of healthy people.[21] Several studies have found increases in the incidence of nonfatal heart disease, including angina and myocardial infarction, among nonsmokers exposed to environmental tobacco smoke.[22,23]

A few small sample cases show direct involvement between environmental tobacco smoke and peripheral vascular disease. For example, Bocanegra and Espinoza[24] reported Raynaud's phenomenon in two successive wives of a chain-smoker. The symptoms of both nonsmokers, as would be expected, subsided after they were no longer exposed to environmental tobacco smoke. Cigarette smoking is a major, preventable risk factor that promotes atherosclerotic peripheral vascular disease,[1,2] and it is likely that environmental tobacco smoke also increases the risk for peripheral vascular disease, although the latter hypothesis remains to be studied.

Mechanisms of Inducing Cardiovascular Disease

Nicotine, the drug in tobacco that causes addiction, produces acute increases in heart rate and blood pressure.[25] Cigarette smoking has been shown to increase platelet aggregation and cause endothelial cell damage.[26–28] Polycyclic aromatic hydrocarbons present in smoke (for example, benzo[a]pyrene) are capable of inducing and accelerating the development of atherosclerosis.[29,30] Exposure to environmental tobacco smoke will also increase carbon monoxide levels in red blood cells. Studies indicate that increased carbon monoxide levels in humans result in a more rapid onset of angina[31] and increased arrhythmias[32] in exercising nonsmokers. A recent study indicates that environmental tobacco smoke sensitizes circulating neutrophils in humans and may cause their subsequent activation and oxidant-mediated tissue damage, leading to carcinogenesis and atherosclerosis.[33] It is likely that these and more yet-to-be-identified mechanisms are involved in increasing the risk of heart disease in persons exposed to environmental tobacco smoke.

Potential for Prevention

Although regulation of tobacco products is specifically prohibited under the Federal Hazardous Substances Act, many actions have been taken to protect the health of nonsmokers. For example, cigarette smoking has been banned from air flights in the 48 contiguous states; and as of March 1991, laws restrict smoking in public places in 46 states, in public-sector workplaces in 38 states, and in private-sector workplaces in 17 states.[34] Many hospitals, health care facilities, and private and public workplaces are smoke-free. The benefit of restricting smoking in buildings and workplaces is obvious, but the effect of a greater awareness of the importance of reducing environmental tobacco smoke in the home has not been evaluated.

The final conclusion of the 1986 Surgeon General's Report was that separating the smokers and nonsmokers within the same air space may reduce but does not eliminate the exposure of nonsmokers to environmental tobacco smoke. Attempts to control tobacco smoke by increasing room ventilation can be futile, and the only sure way to protect nonsmokers from environmental tobacco smoke is to eliminate smoking from areas that they share with nonsmokers. Environmental tobacco smoke must now be considered an environmental toxin from which the public and workers should be protected. Thus, it is the responsibility of the employer to protect workers, and of public building managers, to protect the public from environmental tobacco smoke exposure. It is the responsibility of parents to ensure that their children are not exposed to environmental tobacco smoke in the home, and the responsibility of everyone to eliminate this health hazard from the environment.[1]

Summary

Although the number of cardiovascular deaths associated with environmental tobacco smoke cannot be predicted with absolute certainty, the available evidence indicates that environmental tobacco smoke increases the risk of heart disease. The effects of environmental tobacco smoke on cardiovascular function, platelet function, neutrophil function, and plaque formation are the probable mechanisms leading to heart disease. The risk of death due to heart disease is increased by about 30% among those exposed to environmental tobacco smoke at home and could be much higher in those exposed at the workplace, where higher levels of environmental tobacco smoke may be present. Even though considerable uncertainty is a part of any analysis on the health affects of environmental tobacco smoke because of the difficulty of conducting long-term studies and selecting sample populations, an estimated 35,000–40,000 cardiovascular disease–related deaths and 3,000–5,000 lung cancer deaths due to environmental tobacco smoke exposure have been predicted to occur each year.

The AHA's Council on Cardiopulmonary and Critical Care has concluded that environmental tobacco smoke is a major preventable cause of cardiovascular disease and death. The council strongly supports efforts to eliminate all exposure of nonsmokers to environmental tobacco smoke. This requires that environmental tobacco smoke be treated as an environmental toxin, and ways to protect workers and the public from this health hazard should be developed. According to a 1989 Gallup survey commissioned by the American Lung Association, 86% of nonsmokers think that environmental tobacco smoke is harmful and 77% believe that smokers should abstain in the presence of nonsmokers. However, programs aimed at further educating the public about the cardiovascular effects on nonsmokers of exposure to environmental tobacco smoke must be strengthened and remain a major component of the AHA mission. A smoke-free environment in the home, public buildings, and workplace should be the goal of society.

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PROHIBITING TOBACCO ADVERTISING: APPLYING THE CENTRAL HUDSON GAS TESTS AND POSADAS DE PUERTO RICO

Prior to 1975 commercial speech received no First Amendment Protections. It wasn't until Bigelow v. Virginia, 421 U.S. 809, 95 S.Ct. 2222, 44L. Ed 2d 600 (1975) that the Supreme Court extended limited first amendment protections to commercial speech. Since that time the Court has attempted to refine and define parameters under which commercial speech should and should not be protected. One of the most important Supreme Court cases in refining and defining commercial speech came in 1980. Another came last year.

The tobacco industry and its allies in arguing that a ban on tobacco advertising would be unconstitutional have relied heavily on the application of the four part test set out in Central Hudson Gas and Electric Corp. v. Public Service Commission, 447 US 557, 6SL Ed 2d 341, 100 S.Ct. 2343 (1980). Because the application of these tests go to the heart of the issue of whether a ban on tobacco advertising would be constitutional a careful look at the tests developed in Central Hudson and subsequent discussions and applications in Posadas de Puerto Rico Assoc. v. Tourism Co., 106 S.Ct. 2968 (1986) are warranted.

In Central Hudson, the Court developed a four part test to determine whether or not commercial speech can be restricted. They are:

1) **For commercial speech to come within the protection of the First Amendment, the speech must concern lawful activity and not be misleading.**

2) **The proposed limitations on commercial speech must have a substantial government interest.**

3) **The proposed limitation must directly advance the government's interest.**

4) **The proposed limitation should be no more extensive than is necessary to serve that interest.**

The first test stands on its own and can be considered a threshold question. As the Court said, "...there can be no ... objection ...to the government's suppression of commercial messages that do not accurately inform the public about lawful activity; the government may ban forms of communication more likely to deceive the public than to inform it."

Only if the activity is lawful and not misleading do the other three parts of the Central Hudson tests need to be applied. One could feasibly argue, that all cigarette advertisements are inherently misleading. Women have not "come a long way" by smoking Virginia Slims. Winston's are not "America's Best." Vantage is not the "Taste of Success." Cigarette smoking and tobacco use is the antithesis of healthy, sophisticated, successful, sexually attractive lifestyles.

If, however, the Court were to determine that cigarette advertising was not inherently misleading, the next step is to determine if a ban would meet the other three tests of Central Hudson. Before proceeding to the application of the remaining three tests, it cannot be emphasized enough that the Supreme Court in Posadas has given great deference to the legislature in making the determinations as to whether the government has a substantial interest in limiting the speech, whether the proposed restrictions advance the governments interest, and whether the restrictions are no more extensive than necessary to serve that interest.

The first of the remaining three tests raises the issue of whether or not the government has a substantial interest in the proposed limitations on commercial speech. In the case of tobacco advertising, the answer is obvious.

Even the tobacco industry and its allies would readily and willingly admit that the government has a substantial interest in reducing the incidence of tobacco use in this country. Each year some 350,000 people die smoking related deaths, and another ten million are debilitated. Cigarette smoking costs this nation an estimated $65 billion in health care costs and lost productivity. As the Department of HHS has consistently stated, "cigarette smoking is recognized as the most single preventable cause of death in our society." Thus,

The government's interest is fewer deaths, lower health care costs and

a healthier more productive America

The second part of the three part test requires that the proposed restriction directly advance that government interest. Here the tobacco industry claims that its advertising and promotion affects only brand switching and that advertising does not influence people (particularly young people) to take up the habit. Presently, there are approximately 52 million smokers in this country. Each year the industry loses approximately 2 million of these smokers through death and quitting. Advertising has as its goal, two fundamental objectives - to retain those who already use the product and to encourage those who do not use the product to do so. The advertising strategies for tobacco products are no different (inspite of the industry's claims) than the advertising strategies for other products.[1]

A major target of the industry for market expansion is the youth market. 60% of all new smokers are 14 years old or under and very few people start after the age of 21. Thus the two million-plus smokers the tobacco companies need to recruit each year come predominantly from the under 21 age group. The tobacco industry would have the Congress believe that the relationships between advertising and the use of the product be proven by almost scientific empirical methods. No such elaborate tests or scientific proof is required in the Supreme Court's opinion on the effects of advertising and the use of a product. In both Central Hudson and Posadas the court readily and with very little discussion acknowledged that advertising (regardless of the product) serves to increase demand for the product being advertised. In dealing with this, the Court in Central Hudson stated:

> "The State's interest in energy conservation is directly advanced by the Commission Order at issue here. There is an immediate connection between advertising and demand for electricity. Central Hudson would

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1 Emerson Foote, former chairman of the board of McCann-Erickson, the world's second largest advertising agency, once responsible for $20 million in cigarette accounts, put it this way: "The cigarette industry has been artfully maintaining that cigarette advertising has nothing to do with total sales... This is complete and utter nonsense. The industry knows it is nonsense... I am always amused by the suggestion that advertising, a function that has been shown to increase consumption of virtually every other product, somehow miraculously fails to work for tobacco products." Selling Smoke: Cigarette Advertising and Public Health, Kenneth E. Warner, Ph.D.

> not contest the advertising ban unless it believed that promotion would increase sales."

And in the Posadas the court again applied a "common sense" approach with respect to the affects of advertising on the demand for a product, stating:

> "Step three asks the question whether the challenged restrictions on commercial speech "directly advance" the governments asserted interest. The Puerto Rico Legislature obviously believed when it enacted the advertising restrictions at issue here, that advertising of casino gambling aimed at the residents of Puerto Rico would serve to increase the demand for the product advertised. We think the legislature's belief is a reasonable one, and the fact that appellant has chosen to litigate this case all the way to this Court indicates that appellant shares the legislature's view."

Applying this "common sense" approach used by the Court in Central Hudson and Posadas it is easy to reach a conclusion that cigarette advertising serves to increase and maintain demand for the product. Otherwise, (paraphrasing Central Hudson and Posadas) why else would the industry fight as hard and oppose a ban if advertising didn't promote the use of the product? Some "common sense" examples come to mind. In the 1960's the tobacco industry made a conscience decision to go after the women's market. Through the development of women's cigarettes and an aggressive advertising and promotion campaign, women started smoking more. Smoking rates by teenager girls climbed. Today, lung cancer has replaced breast cancer as the leading cause of death in women. In the 1980's the smokeless tobacco industry began more extensive and aggressive advertising, the result being that use of smokeless products by teenagers rose significantly.

Even the tobacco industry has acknowledged the relationship between advertising and the use of its products. In 1964 the tobacco industry enacted a voluntary code which by their own admission was instituted to ensure that young people were not enticed or seduced into using cigarettes. At hearings before the House Subcommittee on Health and the Environment in 1982 Edward A. Horrigan, Jr. speaking as the Chairman of the Executive Committee of the Tobacco Institute stated:

"Smoking is an adult practice to be considered only by those mature enough to make an informed decision.

In 1963, for example, cigarette companies stopped all advertising and promotional activities in school and college publications and on campus. We also stopped using celebrities and sports figures in advertising.

In 1964, we adopted a cigarette advertising code prohibiting advertising, marketing and sampling directed at young people. Even though the administrative provisions are no longer in effect, each company still adheres to the principles of this code.

In 1969, we offered to cancel all radio and television advertising because of broadcast's unique reach to young people, and in 1971, pursuant to Federal legislation, left the broadcast media."

The truth of the matter is that the very images and techniques that the industry says it doesn't use because they entice people (particularly young people) to use cigarettes are the very images and techniques that the industry repeatedly and consistently uses in all of its advertising and promotion activities. (See attached copy of code).

Clearly then, to remove advertising would be to reduce demand of the product by no longer encouraging smokers to smoke or non smokers to take up the habit. Thus,

The proposed limitation directly advances the government's interest

The third and last part of the three part test requires that the restriction be no more extensive than is necessary to serve the government's interest. Tobacco products as we have stated before, account for over 350,000 deaths each year and debilitate another ten million. Tobacco products are used by a wide spectrum of members of society including a large segment of our nation's youth. It is the official policy of the Department of HHS to reduce the use of tobacco by all segments of the population. In its publication, "The 1990 Health Objectives for the Nation: A Midcourse Review", the Department reiterated the 1990 objectives:

- By 1990, the proportion of adults who smoke should be reduced to below 25 percent

- By 1990 the proportion of women who smoke during pregnancy should be no greater than one half of the proportion of women overall who smoke.

- by 1990 the proportion of children and youth aged 12 to 18 years who smoke should be reduced to below six percent.

Since the release of the 1964 Surgeon General's Report, the public and private sectors have initiated numerous and extensive educational campaigns to discourage the use of tobacco products. The cigarette excise tax was doubled in 1983, making it more costly for young people in particular to buy cigarettes. Disease specific warning labels were required on all tobacco advertisements and packages. But, in spite of all these efforts smoking continues to be a serious public health problem. All of these activities cannot compete with the industry's aggressive and sophisticated $2.3 billion marketing strategy. The industry's own voluntary advertising and promotion code, which as pointed out earlier, was enacted to prevent young people from being enticed into using cigarettes, is regularly violated by the industry. Some of the provisions of the Code state:

- No one depicted in cigarette advertising shall be or appear to be under 25 years of age.

- Cigarette advertising shall not suggest that smoking is essential to social prominence, distinction, success or sexual attraction, nor shall it picture a person smoking in an exaggerated manner.

- Cigarette advertising may picture attractive, healthy looking persons provided there is no suggestion their attractiveness and good health is due to cigarette smoking.

- Cigarette advertising shall not depict as a smoker anyone who is or has been well known as an athlete, nor shall it show any smoker participating in, or obviously just having participated in, a physical

> activity requiring stamina or athletic conditioning beyond that of normal recreation.

Thus, the tobacco industry continues to advertise these addictive, dangerous products as if they are and should be an acceptable and important part of the social environment. As the HHS document clearly pointed out, "for older children and adolescents the ability to resist peer pressure and the declining social acceptance of smoking are key issues. Because there are now more girls and young women smoking than boys and young men, and this attitude appears to be accelerating, efforts to change the smoking attitudes and behaviors of young women also need to be strengthened."

The tobacco industry and its allies would contend that inspite of all these concerted efforts over the last 20 years there are other less restrictive means that should be attempted prior to instituting a ban on advertising and promotion. In determining to what extent other measures need to be attempted, the Supreme Court in Posadas clearly gives the legislature the authority to make such a determination.

The court discussed the specific issue of "counterspeech" as the possibility as a less restrictive means stating:

> "Appellant contends, however, that the First Amendment requires the Puerto Rico Legislature to reduce demand for casino gambling among the residents of Puerto Rico not by suppressing commercial speech that might encourage such gambling, but by promulgating additional speech designed to discourage it. We reject this contention. We think it is up to the legislature to decide whether or not such a "counterspeech" policy would be as effective in reducing the demand for casino gambling as a restriction on advertising. The legislature could conclude, as it apparently did here, that residents of Puerto Rico are already aware of the risks of casino gambling, yet would nevertheless be induced by widespread advertising to engage in such potentially harmful conduct. Cf. Capital Broadcasting Co. v. Mitchell, 333 F. Supp. 582, 585 (DC 1971) (three-judge court) ("Congress had convincing evidence that the Labeling Act of 1965 had not materially reduced the incidence of smoking"), aff'd, 405 U.S. 1000 (1972); Dunagin v. City of Oxford, Miss., 718 F. 2d 738,751 (CA5 1983) (en banc) ("We do not believe that a less restrictive time, place and manner

> restriction, such as a disclaimer warning of the dangers of alcohol, would be effective. The state's concern is not that the public is unaware of the dangers of alcohol.... The concern instead is that advertising will unduly promote alcohol consumption despite known dangers"), cert. denied, 467 U.S. 1259 (1984)." (Emphasis added).

The parallels between the court's rationale in Posadas and tobacco advertising are strikingly similar. The Congress could easily conclude that the residents of the United States are already aware of the risks of cigarette smoking, yet would nevertheless be induced by widespread advertising to engage in such harmful conduct...and that the Congress' concern is not that the public is unaware of the dangers of cigarette smoking....The concern is that advertising will unduly promote cigarette consumption despite known dangers.

The Posadas court further noted:

> "Appellant also makes the related argument that, having chosen to legalize casino gambling for residents of Puerto Rico, the First Amendment prohibits the legislature from using restrictions on advertising to accomplish its goal of reducing demand for such gambling. We disagree. In our view, appellant has the argument backwards. As we noted in the preceding paragraph, it is precisely because the government could have enacted a wholesale prohibition of the underlying conduct that it is permissible for the government to take the less intrusive step of allowing the conduct, but reducing the demand through restrictions on advertising. It would surely be a Pyrrhic victory for casino owners such as appellant to gain recognition of a First Amendment right to advertise their casinos to the residents of Puerto Rico, only to thereby force the legislature into banning casino gambling by residents altogether. It would just as surely be a strange constitutional doctrine which would concede to the legislature the authority to totally ban a product or activity, but deny to the legislature the authority to forbid the stimulation of demand for the product or activity through advertising on behalf of those who would profit from such increased demand. Legislative regulation of products or activities deemed harmful, such as cigarettes, alcoholic beverages, and prostitution, has varied from outright prohibition on the one hand, see, e.g. Cal. Penal Code Ann. Sec. 647(b) (West Supp. 1986) (prohibiting

> soliciting or engaging in acts of prostitution), to legalization of the product or activity with restrictions on stimulation of its demand on the other hand, see, e.g. Nev. Rev. Stat. Secs. 224.345(1), (8) (1986) (authorizing licensing of houses of prostitution except in counties with more than 250,000 population), Secs. 201.430, 201.440 (prohibiting advertising of houses of prostitution "in any public theater, on the public streets of any city or town, or on any public highway," or "in a place of business"). To rule out the latter, intermediate kind of response would require more than we find in the First Amendment."

Thus the Court could easily uphold as constitutional a decision by Congress that,

The proposed limitation is no more extensive than is necessary

The Health Consequences Of Smoking

NICOTINE ADDICTION

a report of the Surgeon General

1988

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Centers for Disease Control
Center for Health Promotion and Education
Office on Smoking and Health
Rockville, Maryland 20857

THE SECRETARY OF HEALTH AND HUMAN SERVICES
WASHINGTON, D.C. 20201

MAY 3 1988

The Honorable George Bush
President of the Senate
Washington, D.C. 20515

Dear Mr. President:

I am pleased to transmit to the Congress the 1987 Surgeon General's Report on the health consequences of smoking, as mandated by Section 8(a) of the Public Health Cigarette Smoking Act of 1969. The Act requires the Secretary of Health and Human Services to transmit an annual report to Congress on the health consequences of smoking and such recommendations for legislation as the Secretary may deem appropriate.

This report, entitled The Health Consequences of Smoking: Nicotine Addiction, examines the scientific evidence that cigarettes and other forms of tobacco are addicting. The issue of tobacco addiction has been addressed in previous Surgeon General's Reports and in the medical literature beginning in the early 1900s. Because of the recent expansion of research in this area, a thorough review of this topic is warranted. Despite the significant health risks of tobacco use outlined in previous reports, many smokers have great difficulty in quitting. This report concludes that such difficulty is in large part due to the addicting properties of nicotine, which is present in all forms of tobacco.

The report further concludes that the processes that determine tobacco addiction are similar to those that determine addiction to other drugs such as heroin and cocaine. Through such understanding, health-care providers may be better able to assist tobacco users in quitting.

Private health organizations, health-care providers, community groups, and government agencies should initiate or strengthen programs to inform the public of the addicting nature of tobacco use. A warning label on the addicting nature of tobacco use should be rotated with other health warnings now required on cigarette and smokeless tobacco packages and advertisements. Preventing the initiation of tobacco use must be a priority because of the difficulty in overcoming nicotine addiction once it is firmly established. Because most cases of nicotine addiction begin during childhood and adolescence, school curricula on the prevention of drug use should also include tobacco.

Cigarette smoking, the chief avoidable cause of premature death in this country, is responsible for more than 300,000 premature deaths each year. The disease impact of smoking justifies placing the problem of tobacco use at the top of the public health agenda. The conclusions of this report provide another compelling reason for strengthening our efforts to reduce tobacco use in our society.

Sincerely,

Otis R. Bowen, M.D.
Secretary

Enclosure

FOREWORD

This 20th Report of the Surgeon General on the health consequences of tobacco use provides an additional important piece of evidence concerning the serious health risks associated with using tobacco.

The subject of this Report, nicotine addiction, was first mentioned in the 1964 Report of the Advisory Committee to the Surgeon General, which referred to tobacco use as "habituating." In the landmark 1979 Report of the Surgeon General, by which time considerably more research had been conducted, smoking was called "the prototypical substance-abuse dependency." Scientists in the field of drug addiction now agree that nicotine, the principal pharmacologic agent that is common to all forms of tobacco, is a powerfully addicting drug.

Recognizing tobacco use as an addiction is critical both for treating the tobacco user and for understanding why people continue to use tobacco despite the known health risks. Nicotine is a psychoactive drug with actions that reinforce the use of tobacco. Efforts to reduce tobacco use in our society must address all the major influences that encourage continued use, including social, psychological, and pharmacologic factors.

After carefully examining the available evidence, this Report concludes that:

- Cigarettes and other forms of tobacco are addicting.
- Nicotine is the drug in tobacco that causes addiction.
- The pharmacologic and behavioral processes that determine tobacco addiction are similar to those that determine addiction to drugs such as heroin and cocaine.

We must recognize both the potential for behavioral and pharmacologic treatment of the addicted tobacco user and the problems of withdrawal. Tobacco use is a disorder which can be remedied through medical attention; therefore, it should be approached by health care providers just as other substance-use disorders are approached: with knowledge, understanding, and persistence. Each health care provider should use every available clinical opportunity to encourage or assist smokers to quit and to help former smokers to maintain abstinence.

To maintain momentum toward a smoke-free society, we also must take steps to prevent young people from beginning to smoke. First, we must insure that every child in every school in this country is educated as to the health risks and the addictive nature of tobacco use. Most jurisdictions require that school curricula include prevention of drug use; therefore, education on the prevention of tobacco use should be included in this effort. Second, warning labels regarding the addictive nature of tobacco use should be required for all tobacco packages and advertisements. Young people in particular may not be aware of the risk of tobacco addiction. Finally, parents and other role models should discourage smoking and other forms of tobacco use among young people. Parents who quit set an example for their children.

Smoking continues to be the chief preventable cause of premature death in this country. Nicotine has addictive properties which help to sustain widespread tobacco use. It is gratifying to see the decline in reported smoking prevalence and cigarette consumption in the United States during the past 25 years. However, we cannot expect to see a sustained decline in rates of smoking-related cancers, cardiovascular disease, and pulmonary disease without sustained public health efforts against tobacco use.

The Public Health Service is committed to preventing tobacco use among youth and to promoting cessation among existing smokers. We hope that this Report will assist the health care community, voluntary health agencies, and our Nation's schools in working with us to reduce tobacco use in our society.

Robert E. Windom, M.D.
Assistant Secretary for Health

PREFACE

This Report of the Surgeon General is the U.S. Public Health Service's 20th Report on the health consequences of tobacco use and the 7th issued during my tenure as Surgeon General. Eighteen Reports have been released previously as part of the health consequences of smoking series; a report on the health consequences of using smokeless tobacco was released in 1986.

Previous Reports have reviewed the medical and scientific evidence establishing the health effects of cigarette smoking and other forms of tobacco use. Tens of thousands of studies have documented that smoking causes lung cancer, other cancers, chronic obstructive lung disease, heart disease, complications of pregnancy, and several other adverse health effects.

Epidemiologic studies have shown that cigarette smoking is responsible for more than 300,000 deaths each year in the United States. As I stated in the Preface to the 1982 Surgeon General's Report, smoking is the chief avoidable cause of death in our society.

From 1964 through 1979, each Surgeon General's Report addressed the major health effects of smoking. The 1979 Report provided the most comprehensive review of these effects. Following the 1979 Report, each subsequent Report has focused on specific populations (women in 1980, workers in 1985), specific diseases (cancer in 1982, cardiovascular disease in 1983, chronic obstructive lung disease in 1984), and specific topics (low-tar, low-nicotine cigarettes in 1981, involuntary smoking in 1986).

This Report explores in great detail another specific topic: nicotine addiction. Careful examination of the data makes it clear that cigarettes and other forms of tobacco are addicting. An extensive body of research has shown that nicotine is the drug in tobacco that causes addiction. Moreover, the processes that determine tobacco addiction are similar to those that determine addiction to drugs such as heroin and cocaine.

Actions of Nicotine

All tobacco products contain substantial amounts of nicotine. Nicotine is absorbed readily from tobacco smoke in the lungs and from smokeless tobacco in the mouth or nose. Levels of nicotine in

the blood are similar in magnitude in people using different forms of tobacco. Once in the blood stream, nicotine is rapidly distributed throughout the body.

Nicotine is a powerful pharmacologic agent that acts in a variety of ways at different sites in the body. After reaching the blood stream, nicotine enters the brain, interacts with specific receptors in brain tissue, and initiates metabolic and electrical activity in the brain. In addition, nicotine causes skeletal muscle relaxation and has cardiovascular and endocrine (i.e., hormonal) effects.

Human and animal studies have shown that nicotine is the agent in tobacco that leads to addiction. The diversity and strength of its actions on the body are consistent with its role in causing addiction.

Tobacco Use as an Addiction

Standard definitions of drug addiction have been adopted by various organizations including the World Health Organization and the American Psychiatric Association. Although these definitions are not identical, they have in common several criteria for establishing a drug as addicting.

The central element among all forms of drug addiction is that the user's behavior is largely controlled by a psychoactive substance (i.e., a substance that produces transient alterations in mood that are primarily mediated by effects in the brain). There is often compulsive use of the drug despite damage to the individual or to society, and drug-seeking behavior can take precedence over other important priorities. The drug is "reinforcing"—that is, the pharmacologic activity of the drug is sufficiently rewarding to maintain self-administration. "Tolerance" is another aspect of drug addiction whereby a given dose of a drug produces less effect or increasing doses are required to achieve a specified intensity of response. Physical dependence on the drug can also occur, and is characterized by a withdrawal syndrome that usually accompanies drug abstinence. After cessation of drug use, there is a strong tendency to relapse.

This Report demonstrates in detail that tobacco use and nicotine in particular meet all these criteria. The evidence for these findings is derived from animal studies as well as human observations. Leading national and international organizations, including the World Health Organization and the American Psychiatric Association, have recognized chronic tobacco use as a drug addiction.

Some people may have difficulty in accepting the notion that tobacco is addicting because it is a legal product. The word "addiction" is strongly associated with illegal drugs such as cocaine and heroin. However, as this Report shows, the processes that

determine tobacco addiction are similar to those that determine addiction to other drugs, including illegal drugs.

In addition, some smokers may not believe that tobacco is addicting because of a reluctance to admit that one's behavior is largely controlled by a drug. On the other hand, most smokers admit that they would like to quit but have been unable to do so. Smokers who have repeatedly failed in their attempts to quit probably realize that smoking is more than just a simple habit.

Many smokers have quit on their own ("spontaneous remission") and some smokers smoke only occasionally. However, spontaneous remission and occasional use also occur with the illicit drugs of addiction, and in no way disqualify a drug from being classified as addicting. Most narcotics users, for example, never progress beyond occasional use, and of those who do, approximately 30 percent spontaneously remit. Moreover, it seems plausible that spontaneous remitters are largely those who have either learned to deliver effective treatments to themselves or for whom environmental circumstances have fortuitously changed in such a way as to support drug cessation and abstinence.

Treatment

Like other addictions, tobacco use can be effectively treated. A wide variety of behavioral interventions have been used for many years, including aversion procedures (e.g., satiation, rapid smoking), relaxation training, coping skills training, stimulus control, and nicotine fading. In recognition of the important role that nicotine plays in maintaining tobacco use, nicotine replacement therapy is now available. Nicotine polacrilex gum has been shown in controlled trials to relieve withdrawal symptoms. In addition, some (but not all) studies have shown that nicotine gum, as an adjunct to behavioral interventions, increases smoking abstinence rates. In recent years, multicomponent interventions have been applied successfully to the treatment of tobacco addiction.

Public Health Strategies

The conclusion that cigarettes and other forms of tobacco are addicting has important implications for health professionals, educators, and policy-makers. In treating the tobacco user, health professionals must address the tenacious hold that nicotine has on the body. More effective interventions must be developed to counteract both the psychological and pharmacologic addictions that accompany tobacco use. More research is needed to evaluate how best to treat those with the strongest dependence on the drug. Treatment of tobacco addiction should be more widely available and should be

considered at least as favorably by third-party payors as treatment of alcoholism and illicit drug addiction.

The challenge to health professionals is complicated by the array of new nicotine delivery systems that are being developed and introduced in the marketplace. Some of these products are produced by tobacco manufacturers; others may be marketed as devices to aid in smoking cessation. These new products may be more toxic and more addicting than the products currently on the market. New nicotine delivery systems should be evaluated for their toxic and addictive effects; products intended for use in smoking cessation also should be evaluated for efficacy.

Public information campaigns should be developed to increase community awareness of the addictive nature of tobacco use. A health warning on addiction should be rotated with the other warnings now required on cigarette and smokeless tobacco packages and advertisements. Prevention of tobacco use should be included along with prevention of illicit drug use in comprehensive school health education curricula. Many children and adolescents who are experimenting with cigarettes and other forms of tobacco state that they do not intend to use tobacco in later years. They are unaware of, or underestimate, the strength of tobacco addiction. Because this addiction almost always begins during childhood or adolescence, children need to be warned as early as possible, and repeatedly warned through their teenage years, about the dangers of exposing themselves to nicotine.

This Report shows conclusively that cigarettes and other forms of tobacco are addicting in the same sense as are drugs such as heroin and cocaine. Most adults view illegal drugs with scorn and express disapproval (if not outrage) at their sale and use. This Nation has mobilized enormous resources to wage a war on drugs — illicit drugs. We should also give priority to the one addiction that is killing more than 300,000 Americans each year.

We as citizens, in concert with our elected officials, civic leaders, and public health officers, should establish appropriate public policies for how tobacco products are sold and distributed in our society. With the evidence that tobacco is addicting, is it appropriate for tobacco products to be sold through vending machines, which are easily accessible to children? Is it appropriate for free samples of tobacco products to be sent through the mail or distributed on public property, where verification of age is difficult if not impossible? Should the sale of tobacco be treated less seriously than the sale of alcoholic beverages, for which a specific license is required (and revoked for repeated sales to minors)?

In the face of overwhelming evidence that tobacco is addicting, policy-makers should address these questions without delay. To

achieve our goal of a smoke-free society, we must give this problem the serious attention it deserves.

C. Everett Koop, M.D., Sc.D.
Surgeon General

CHAPTER I

INTRODUCTION, OVERVIEW, SUMMARY, AND CONCLUSIONS

Introduction

Development and Organization of this Report

This Report was developed by the Office on Smoking and Health, Center for Health Promotion and Education, Centers for Disease Control, Public Health Service of the U.S. Department of Health and Human Services as part of the Department's responsibility, under Public Law 91-222, to report new and current information on smoking and health to the United States Congress.

The scientific content of this Report reflects the contributions of more than 50 scientists representing a wide variety of relevant disciplines. These experts, known for their understanding of and work in specific content areas, prepared manuscripts for incorporation into this Report. The Office on Smoking and Health and its consultants edited and consolidated the individual manuscripts into appropriate chapters. These draft chapters were subjected to an extensive outside peer review (see Acknowledgments for individuals and their affiliations) whereby each chapter was reviewed by up to 11 experts. Based on the comments of these reviewers, the chapters were revised and the entire volume was assembled. This revised edition of the Report was resubjected to review by 20 distinguished scientists inside and outside the Federal Government, both in this country and abroad. Parallel to this review, the entire Report was also submitted for review to 12 institutes and agencies within the U.S. Public Health Service. The comments from the senior scientific reviewers and the agencies were used to prepare the final volume of this Report.

This Report contains a Foreword by the Assistant Secretary for Health, a Preface by the Surgeon General of the U.S. Public Health Service, and the following chapters and appendices:

Chapter I. Introduction, Overview, Summary, and Conclusions
Chapter II. Nicotine: Pharmacokinetics, Metabolism, and Pharmacodynamics
Chapter III. Nicotine: Sites and Mechanisms of Actions
Chapter IV. Tobacco Use as Drug Dependence
Chapter V. Tobacco Use Compared to Other Drug Dependencies
Chapter VI. Effects of Nicotine That May Promote Tobacco Use
Chapter VII. Treatment of Tobacco Dependence
Appendix A. Trends in Tobacco Use in the United States
Appendix B. Toxicity of Nicotine

Overview

This Report of the Surgeon General on tobacco and health focuses on the pharmacologic basis of tobacco addiction. Previous Surgeon General's Reports have reviewed the medical and scientific evidence establishing that cigarette smoking and tobacco use in other forms are deleterious to health. Several reports emphasized particular diseases (e.g., 1982 Report on cancer (US DHHS 1982), 1983 Report on cardiovascular disease (US DHHS 1983a), 1984 Report on chronic obstructive lung disease (US DHHS 1984a)); some reports concentrated on specific populations (e.g., 1980 Report on women (US DHHS 1980)); and some reports dealt with particular aspects of smoking (e.g., 1986 Report on involuntary smoking (US DHHS 1986a)). These reports have been important because so many individuals engage in a behavior that causes morbidity and premature mortality.

The present Report addresses a central issue of the tobacco and health problem: Why do people smoke and in other ways consume tobacco products? Specifically, this Report reviews the pharmacologic basis of the disease-producing and life-threatening behavior of tobacco use. Psychological and social factors are also important influences on tobacco use, but a detailed review of these factors is beyond the scope of this Report. Reviews of this literature include previous reports of the Surgeon General (US DHEW 1979; US DHHS 1980, 1982, 1983a, 1984a), research monographs from the National Institute on Drug Abuse (NIDA) (Jarvik et al. 1977; Krasnegor 1978, 1979a,b,c; Grabowski and Bell 1983), and articles by scientists who study tobacco use and nicotine (Russell 1971, 1976; Gritz 1980; Henningfield 1984).

This Report reviews evidence that tobacco use is addicting and that nicotine is the active pharmacologic agent of tobacco that causes this addictive behavior. Previous Surgeon General's Reports have focused on evidence that cigarette smoking and tobacco use are health hazards. Now that those relationships are well-documented and well-known, this Report addresses addictive properties of cigarette smoking and tobacco use in order to help develop more effective prevention and cessation programs.

This Report topic is particularly timely because of recent advances and extensive data gathered in the 1980s relevant to the issue of tobacco addiction. Since the early 1900s scientific literature and historical anecdotes have provided evidence that tobacco use is a form of drug addiction. In the 1970s, however, research efforts increased considerably on various aspects of tobacco addiction, including nicotine pharmacokinetics, pharmacodynamics, self-administration, withdrawal, dependence, and tolerance. In addition, advances in the neurosciences have begun to reveal effects of nicotine in the brain and body that may help to explain why tobacco use is reinforcing and difficult to give up. These issues are addressed

in this Report. Finally, recent developments in the use of nicotine replacement in smoking cessation emphasize the importance of pharmacologic aspects of cigarette smoking.

Concepts of drug addiction or drug dependence are discussed in detail in Chapters IV and V. It is useful to begin this Report with a brief summary of main points about drug dependence that provide the foundation for the findings of the Report.

The terms "drug addiction" and "drug dependence" are scientifically equivalent: both terms refer to the behavior of repetitively ingesting mood-altering substances by individuals. The term "drug dependence" has been increasingly adopted in the scientific and medical literature as a more technical term, whereas the term "drug addiction" continues to be used by NIDA and other organizations when it is important to provide information at a more general level. Throughout this Report, both terms are used and they are used synonymously.

The main conclusions of the Report are based upon concepts of drug dependence that have been developed by expert committees of the World Health Organization, as well as in publications of NIDA and the American Psychiatric Association. These concepts were used to develop a set of criteria to determine whether tobacco-delivered nicotine is addicting. The criteria for drug dependence include primary and additional indices and are summarized below.

CRITERIA FOR DRUG DEPENDENCE

Primary Criteria

- Highly controlled or compulsive use
- Psychoactive effects
- Drug-reinforced behavior

Additional Criteria

- Addictive behavior often involves:
  - -stereotypic patterns of use
  - -use despite harmful effects
  - -relapse following abstinence
  - -recurrent drug cravings
- Dependence-producing drugs often produce:
  - -tolerance
  - -physical dependence
  - -pleasant (euphoriant) effects

The primary criteria listed above are sufficient to define drug dependence. Highly controlled or compulsive use indicates that drug-

seeking and drug-taking behavior is driven by strong, often irresistible urges. It can persist despite a desire to quit or even repeated attempts to quit. Such behavior is also referred to as "habitual" behavior. To distinguish drug dependence from habitual behaviors not involving drugs, it must be demonstrated that a drug with psychoactive (mood-altering) effects in the brain enters the blood stream. Furthermore, drug dependence is defined by the occurrence of drug-motivated behavior; therefore, the psychoactive chemical must be capable of functioning as a reinforcer that can directly strengthen behavior leading to further drug ingestion.

Additional criteria are often used to help characterize drug dependence. Several are associated with the drug-taking behavior itself: (1) the behavior may develop into regular temporal and physical patterns of use (repetitive and stereotypic); (2) drug use may persist despite adverse physical, psychological, or social consequences; (3) quitting episodes are often followed by resumption of drug use (relapse); (4) urges (cravings) to use the drug may be recurrent and persistent, especially during drug abstinence. Similarly, several common effects of dependence-producing drugs can strengthen their control over behavior and increase the likelihood of harm by contributing to the regularity and overall level of drug intake: (1) diminished responsiveness (tolerance) to the effects of a drug occurs, and may be accompanied by increased intake over time; (2) abstinence-associated withdrawal reactions (due to physical dependence) can motivate further drug intake; (3) effects that are considered pleasant (euphoriant) to the drug user can be provided by the drug itself. Dependence-producing drugs can also produce effects that individuals find useful. For example, many addicting drugs have therapeutic uses in medical treatments of various disorders. Most medically approved drugs that are addicting, however, are generally only available by prescription. Effects of a drug considered by the individual to be useful can promote initiation of drug use, strengthen the addiction, and contribute to relapse following cessation of use.

Tobacco and nicotine are considered in the Report in light of the above criteria. In brief, the organization of the Report is as follows: review of evidence that tobacco use is accompanied by orderly patterns of uptake of nicotine in the body and brain resulting in the development of tolerance (Chapter II); review of how effects of nicotine in the brain and the rest of the body are chemically mediated (Chapter III); review of the evidence that tobacco is addicting and that nicotine is an addicting drug (Chapter IV); comparison of tobacco use with other addictions and of nicotine with other addicting drugs (Chapter V); review of possible effects of nicotine that may promote the use of tobacco and present impediments to quitting smoking (Chapter VI); review of strategies for

helping people to achieve and maintain tobacco abstinence (Chapter VII). In addition, appendices are included that summarize information regarding trends in tobacco use (Appendix A) and information regarding the toxicity of nicotine itself (Appendix B). A summary of the main findings of the Report follows.

Major Conclusions

1. **Cigarettes and other forms of tobacco are addicting.**

2. **Nicotine is the drug in tobacco that causes addiction.**

3. **The pharmacologic and behavioral processes that determine tobacco addiction are similar to those that determine addiction to drugs such as heroin and cocaine.**

Brief History Relevant to this Report

Tobacco products have been used for centuries. The tobacco plant was native to the New World. The oldest cited evidence of tobacco use appears on a Mayan stone carving dated from 600 to 900 A.D. There are reports of tobacco smoking in Christopher Columbus' diary in 1492; reports of tobacco smoking appear in the logs of other European explorers of the New World in the 16th century. Since the colonial period, tobacco has been an integral part of the American economy (Robert 1949).

Tobacco use permeated the New World and quickly spread throughout the rest of the world during the 16th and 17th centuries. As use of tobacco products spread, so did controversy over the effects of these products. Throughout history, while some persons extolled the virtues of tobacco (including numerous alleged medicinal uses), others condemned its use. George Washington is attributed with exhorting the home front during the Revolutionary War, "If you can't send money, send tobacco." In contrast, Dr. Benjamin Rush condemned tobacco use in his 1798 book *Essays*. The controversy continued into the 19th century with no convincing scientific or medical evidence to support either position (Robert 1949).

In 1856–57 the British medical journal *Lancet* published opinions of 50 physicians on tobacco use. Many opponents attributed increased crime, nervous paralysis, loss of intellectual abilities, and visual impairment to tobacco use—all of these claims lacked convincing evidence. In restating the main arguments of the tobacco proponents, the *Lancet* editors wrote that tobacco use "...must have some good or at least pleasurable effects; that, if its evil effects were

so dreadful as stated the human race would have ceased to exist" (Lancet 1857).

While the health-promoting and health-damaging effects of tobacco products were being debated throughout the 17th and 18th centuries, scientists were trying to determine the chief active ingredient in tobacco. In the early 1800s the oily essence of tobacco was discovered by Cerioli and by Vauquelin. This active substance was named "Nicotianine," after Jean Nicot, who sent tobacco seeds from Portugal to the French court at the end of the 16th century. In 1828, Posselt and Reimann at the University of Heidelberg isolated the pure form of Nicotianine and renamed it "Nikotin." The chemical's empirical formula, $C_{10}H_{14}N_2$, was determined in the 1840s, and "nicotine" was synthesized in the 1890s (Robert 1949).

Since the late 1800s, research on the pharmacologic actions of nicotine has contributed substantially to basic information about the nervous system (Kharkevich 1980; Volle 1980). The classic work by Langley and Dickinson (1889) on nicotine's effects in autonomic ganglia led to the postulates that chemicals transmit information between neurons and that there are receptors on cells that respond functionally to stimulation by specific chemicals. As early as the 1920s and 1930s, some investigators were concluding that nicotine was responsible for the compulsive use of tobacco products (Armstrong-Jones 1927; Dorsey 1936; Lewin 1931). Johnston (1942) concluded that, "smoking tobacco is essentially a means of administering nicotine, just as smoking opium is a means of administering morphine."

Throughout the 20th century, research has continued to investigate the role of nicotine in tobacco use. The 1964 Report of the Surgeon General's Advisory Committee on Smoking and Health (US PHS 1964) held that: "The habitual use of tobacco is related primarily to psychological and social drives, reinforced and perpetuated by the pharmacologic actions of nicotine on the central nervous system. Nicotine-free tobacco or other plant materials do not satisfy the needs of those who acquire the tobacco habit." The 1964 Report, relying upon a distinction (that is no longer made) between "habituating" and "addicting" drugs, asserted that tobacco was habituating and not addicting. The distinction in 1964 between habituating drugs (including cocaine and amphetamines) and addicting drugs (including opiates and barbiturates) was based on: (1) whether the drug produced clear physical dependence; (2) whether damage was mainly to the individual user (habituating drugs) or to society (addicting drugs); and (3) the strength of the habitual behavior that developed. There was no question at the time of the 1964 Report that nicotine was the critical pharmacologic agent for tobacco use, but its role was then considered to be more similar to cocaine and amphetamines than to opiates and barbiturates. Later

in 1964 the World Health Organization dropped this semantic distinction between habituating and addicting drugs because it was recognized that habitual use could be as strongly developed for cocaine as for morphine, that social damage generally accompanied personal damage, and that behavioral characteristics of drug use could be similar for the so-called habituating and addicting drugs. In an effort to shift the focus to dependent patterns of behavior and away from moral and social issues associated with the term addiction, the term dependence was recommended.

It is now clear that even by the earlier distinction in nomenclature, cigarettes and other forms of tobacco are addicting and actions of nicotine provide the pharmacologic basis of tobacco addiction. The term "dependence producing" may also be used to describe cigarettes and other forms of tobacco use, analogous to actions of other drugs (e.g., opiates, cocaine). Since 1964, considerable additional evidence has been compiled that substantiates these conclusions. The present Report reviews this information and the relevant literature.

Previous Surgeon General's Reports provided current reviews of the health consequences of cigarette smoking particularly relevant to public health. For example, despite the accumulating evidence, in the early 1960s there was little recognition by the public of the health hazards of smoking. Each Report examined specific information considered to be important for public dissemination. A brief review of topics addressed in these reports provides the background for the present Report.

In the late 1950s, the U.S. Public Health Service, the National Cancer Institute, the National Heart Institute, the American Cancer Society, and the American Heart Association appointed a study group to examine the available evidence on smoking and health. This study group concluded that excessive cigarette smoking is a causative factor in lung cancer.

In 1962, Surgeon General Luther Terry established an advisory committee on smoking and health. This committee released its Report on January 11, 1964, concluding that cigarette smoking is a cause of lung cancer in men and a suspected cause of lung cancer in women, and increased the risk of dying from pulmonary emphysema. The next Report was issued in 1967 (US PHS 1968a) and stated that "the case for cigarette smoking as the principal cause of lung cancer is overwhelming." Further, the 1967 Report concluded that: "There is an increasing convergence of many types of evidence . . . which strongly suggests that cigarette smoking can cause death from coronary heart disease." The 1967 Report also concluded that "Cigarette smoking is the most important of the causes of chronic non-neoplastic bronchopulmonary disease in the United States."

The 1968 and 1969 Reports (US PHS 1968b, 1969) strengthened the conclusions reached in 1967. The 1971 Report provided a detailed

review of the evidence to date regarding health consequences of smoking (US DHEW 1971). The subsequent reports (1972 to 1976) continued to review the increasing evidence associating cigarette smoking with many health hazards. The 1972 Report also discussed involuntary or passive smoking (US DHEW 1972). The 1973 Report included some data on the health hazards of smoking pipes and cigars (US PHS 1973). The 1975 Report updated information on the health effects of involuntary or passive smoking (US DHEW 1975). The combined 1977–78 Report discussed smoking-related problems unique to women (US DHEW 1978).

At the time of its release, the 1979 Report was the most comprehensive review by a Surgeon General's Report of the health consequences of smoking, smoking behavior, and smoking control. In addition to providing a thorough review of the health consequences of smoking, the 1979 Report discussed the health consequences of using forms of tobacco other than cigarettes (pipes, cigars, and smokeless tobacco). Moreover, the 1979 Report expanded the scope of the previous reports and examined behavioral, pharmacologic, and social factors influencing the initiation, maintenance, and cessation of cigarette smoking. Relevant to the topic of the present Report, the 1979 Report concluded that "it is no exaggeration to say that smoking is the prototypical substance-abuse dependency and that improved knowledge of this process holds great promise for prevention of risk." Since the release of the 1979 Report, each subsequent Report has focused on a specific population or setting (women in 1980 (US DHHS 1980), the workplace in 1985 (US DHHS 1985)), a specific topic (health effects of low-tar and low-nicotine cigarettes in 1981 (US DHHS 1981), involuntary smoking in 1986 (US DHHS 1986a)), or a specific disease (cancer in 1982 (US DHHS 1982), cardiovascular diseases in 1983 (US DHHS 1983a), chronic obstructive lung disease in 1984 (US DHHS 1984a)).

In addition to the previous Surgeon General's Reports, several other developments and publications provide relevant background for the present Report. For example, numerous monographs prepared in the 1970s by the National Institute on Drug Abuse (NIDA) considered tobacco use as a form of drug dependence. In 1980, the American Psychiatric Association, in its Diagnostic and Statistical Manual of Mental Disorders, included tobacco dependence as a substance abuse disorder and tobacco withdrawal as an organic mental disorder (APA 1980). The 1987 revised edition of this manual (APA 1987), in recognition of the role of nicotine, changed "tobacco withdrawal" to "nicotine withdrawal." In 1982, the Director of NIDA testified to Congress that the position of NIDA was that tobacco use could lead to dependence and that nicotine was a prototypic dependence-producing drug. In a 1983 publication, "Why People Smoke Cigarettes," the U.S. Public Health Service supported this

position of NIDA regarding tobacco and nicotine (US DHHS 1983b). In the 1984 NIDA Triennial Report to Congress, nicotine was labeled a prototypic dependence-producing drug and the role of nicotine in tobacco use was considered to be analogous to the roles of morphine, cocaine, and ethanol, in the use of opium, coca-derived products, and alcoholic beverages, respectively (US DHHS 1984b). In 1986, a consensus conference of the National Institutes of Health and the Report of the Advisory Committee to the Surgeon General on the health consequences of using smokeless tobacco concluded that smokeless tobacco can be addicting and that nicotine is a dependence-producing (i.e., addicting) drug (US DHHS 1986b).

The present Report is the 20th such report issued by the Public Health Service on the health consequences of tobacco use. The deleterious effects of cigarette smoking are now well known. Therefore, this Report focuses on pharmacologic information to help understand why people smoke. Such information will assist health professionals in developing effective strategies to prevent initiation and to promote cessation. The literature reviewed in this Report indicates that tobacco use is an addictive behavior. It is the purpose of this Report to thoroughly review the relevant literature.

Chapter Conclusions

In addition to the three overall conclusions of this Report, there are many other substantive conclusions. These points are listed under the appropriate Chapter and Appendix headings.

Chapter II: Nicotine: Pharmacokinetics, Metabolism, and Pharmacodynamics

1. All tobacco products contain substantial amounts of nicotine and other alkaloids. Tobaccos from low-yield and high-yield cigarettes contain similar amounts of nicotine.
2. Nicotine is absorbed readily from tobacco smoke in the lungs and from smokeless tobacco in the mouth or nose. Levels of nicotine in the blood are similar in magnitude in people using different forms of tobacco. With regular use, levels of nicotine accumulate in the body during the day and persist overnight. Thus, daily tobacco users are exposed to the effects of nicotine for 24 hr each day.
3. Nicotine that enters the blood is rapidly distributed to the brain. As a result, effects of nicotine on the central nervous system occur rapidly after a puff of cigarette smoke or after absorption of nicotine from other routes of administration.
4. Acute and chronic tolerance develops to many effects of nicotine. Such tolerance is consistent with reports that initial

use of tobacco products, such as in adolescents first beginning to smoke, is usually accompanied by a number of unpleasant symptoms which disappear following chronic tobacco use.

Chapter III: Nicotine: Sites and Mechanisms of Actions

1. Nicotine is a powerful pharmacologic agent that acts in the brain and throughout the body. Actions include electrocortical activation, skeletal muscle relaxation, and cardiovascular and endocrine effects. The many biochemical and electrocortical effects of nicotine may act in concert to reinforce tobacco use.
2. Nicotine acts on specific binding sites or receptors throughout the nervous system. Nicotine readily crosses the blood–brain barrier and accumulates in the brain shortly after it enters the body. Once in the brain, it interacts with specific receptors and alters brain energy metabolism in a pattern consistent with the distribution of specific binding sites for the drug.
3. Nicotine and smoking exert effects on nearly all components of the endocrine and neuroendocrine systems (including catecholamines, serotonin, corticosteroids, pituitary hormones). Some of these endocrine effects are mediated by actions of nicotine on brain neurotransmitter systems (e.g., hypothalamic–pituitary axis). In addition, nicotine has direct peripherally mediated effects (e.g., on the adrenal medulla and the adrenal cortex).

Chapter IV: Tobacco Use as Drug Dependence

1. Cigarettes and other forms of tobacco are addicting. Patterns of tobacco use are regular and compulsive, and a withdrawal syndrome usually accompanies tobacco abstinence.
2. Nicotine is the drug in tobacco that causes addiction. Specifically, nicotine is psychoactive ("mood altering") and can provide pleasurable effects. Nicotine can serve as a reinforcer to motivate tobacco-seeking and tobacco-using behavior. Tolerance develops to actions of nicotine such that repeated use results in diminished effects and can be accompanied by increased intake. Nicotine also causes physical dependence characterized by a withdrawal syndrome that usually accompanies nicotine abstinence.
3. The physical characteristics of nicotine delivery systems can affect their toxicity and addictiveness. Therefore, new nicotine delivery systems should be evaluated for their toxic and addictive effects.

Chapter V: Tobacco Use Compared to Other Drug Dependencies

1. The pharmacologic and behavioral processes that determine tobacco addiction are similar to those that determine addiction to drugs such as heroin and cocaine.
2. Environmental factors including drug-associated stimuli and social pressure are important influences of initiation, patterns of use, quitting, and relapse to use of opioids, alcohol, nicotine, and other addicting drugs.
3. Many persons dependent upon opioids, alcohol, nicotine, or other drugs are able to give up their drug use outside the context of treatment programs; other persons, however, require the assistance of formal cessation programs to achieve lasting drug abstinence.
4. Relapse to drug use often occurs among persons who have achieved abstinence from opioids, alcohol, nicotine, or other drugs.
5. Behavioral and pharmacologic intervention techniques with demonstrated efficacy are available for the treatment of addiction to opioids, alcohol, nicotine, and other drugs.

Chapter VI: Effects of Nicotine That May Promote Tobacco Dependence

1. After smoking cigarettes or receiving nicotine, smokers perform better on some cognitive tasks (including sustained attention and selective attention) than they do when deprived of cigarettes or nicotine. However, smoking and nicotine do not improve general learning.
2. Stress increases cigarette consumption among smokers. Further, stress has been identified as a risk factor for initiation of smoking in adolescence.
3. In general, cigarette smokers weigh less (approximately 7 lb less on average) than nonsmokers. Many smokers who quit smoking gain weight.
4. Food intake and probably metabolic factors are involved in the inverse relationship between smoking and body weight. There is evidence that nicotine plays an important role in the relationship between smoking and body weight.

Chapter VII: Treatment of Tobacco Dependence

1. Tobacco dependence can be treated successfully.
2. Effective interventions include behavioral approaches alone and behavioral approaches with adjunctive pharmacologic treatment.

3. Behavioral interventions are most effective when they include multiple components (procedures such as aversive smoking, skills training, group support, and self-reward). Inclusion of too many treatment procedures can lead to less successful outcome.
4. Nicotine replacement can reduce tobacco withdrawal symptoms and may enhance the efficacy of behavioral treatment.

Appendix A: Trends in Tobacco Use in the United States

1. An estimated 32.7 percent of men and 28.3 percent of women smoked cigarettes regularly in 1985. The overall prevalence of smoking in the United States decreased from 36.7 percent in 1976 (52.4 million adults) to 30.4 percent in 1985 (51.1 million adults).
2. In 1985, the mean reported number of cigarettes smoked per day was 21.8 for male smokers and 18.1 for female smokers.
3. Smoking is more common in lower socioeconomic categories (blue-collar workers or unemployed persons, less educated persons, and lower income groups) than in higher socioeconomic categories. For example, the prevalence of smoking in 1985 among persons without a high school diploma was 35.4 percent, compared with 16.5 percent among persons with postgraduate college education.
4. An estimated 18.7 percent of high school seniors reported daily use of cigarettes in 1986. The prevalence of daily use of one or more cigarettes among high school seniors declined between 1975 and 1986 by approximately 35 percent. Most of the decline occurred between 1977 and 1981. Since 1976, the smoking prevalence among females has consistently been slightly higher than among males.
5. The use of cigars and pipes has declined 80 percent since 1964.
6. Smokeless tobacco use has increased substantially among young men and has declined among older men since 1975. An estimated 8.2 percent of 17- to 19-year-old men were users of smokeless tobacco products in 1986.

Appendix B: Toxicity of Nicotine

1. At high exposure levels, nicotine is a potent and potentially lethal poison. Human poisonings occur primarily as a result of accidental ingestion or skin contact with nicotine-containing insecticides or, in children, after ingestion of tobacco or tobacco juices.
2. Mild nicotine intoxication occurs in first-time smokers, nonsmoking workers who harvest tobacco leaves, and people who

chew excessive amounts of nicotine polacrilex gum. Tolerance to these effects develops rapidly.

3. Nicotine exposure in long-term tobacco users is substantial, affecting many organ systems (Chapters II and III). Pharmacologic actions of nicotine may contribute to the pathogenesis of smoking-related diseases, although direct causation has not yet been determined. Of particular concern are cardiovascular disease, complications of hypertension, reproductive disorders, cancer, and gastrointestinal disorders, including peptic ulcer disease and gastroesophageal reflux.
4. The risks of short-term nicotine replacement therapy as an aid to smoking cessation in healthy people are acceptable and substantially outweighed by the risks of cigarette smoking.

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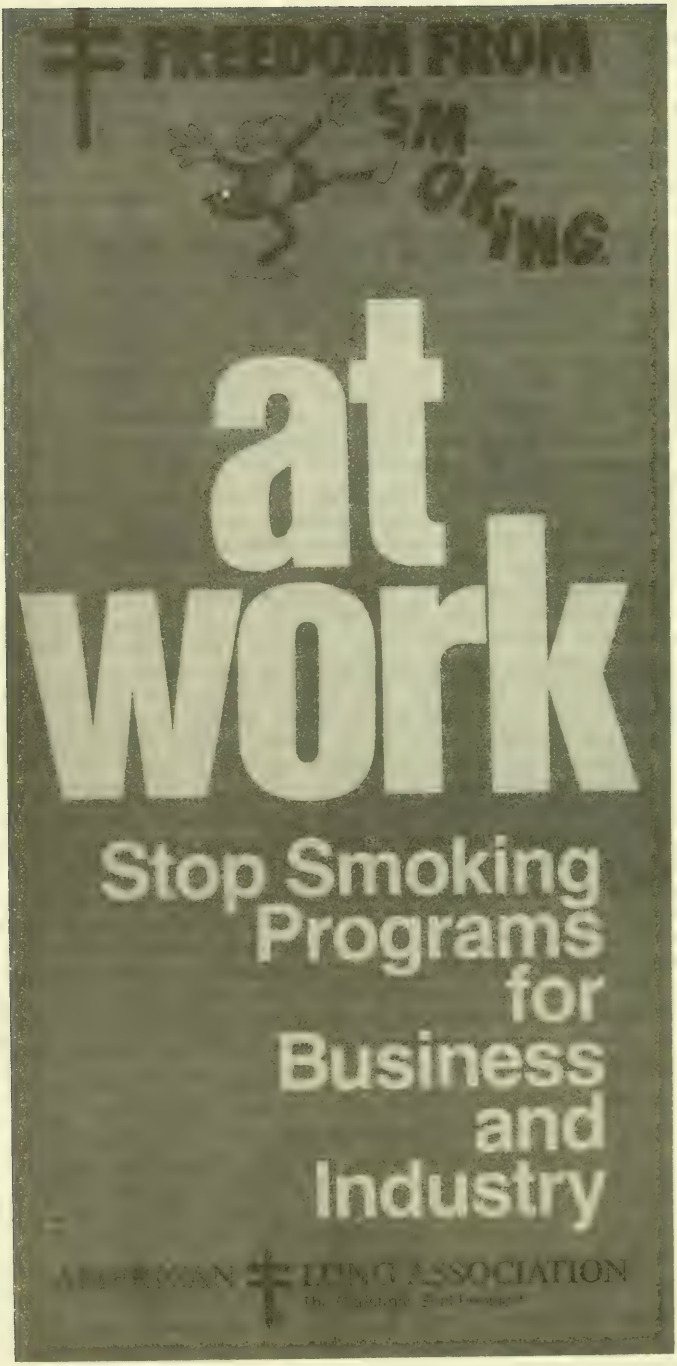
FREEDOM FROM
SMOKING
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work
Stop Smoking
Programs
for
Business
and
Industry
AMERICAN LUNG ASSOCIATION

- The costs of smoking at work—in health terms as well as financial ones—are skyrocketing. The issue concerns employers and employees, smokers and nonsmokers.

- Your local American Lung Association has nationally tested programs to help smokers stop smoking permanently. And your Lung Association can help your company develop a policy to protect nonsmokers on the job.

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SMOKING AT THE WORKPLACE

Both employees and employers are increasingly concerned about the issue of smoking at the workplace. For good reason.

SMOKERS WANT TO QUIT

Smokers themselves are worried about their life-destroying habit.

- Studies show 9 out of 10 smokers say they would quit if they could find a way that works.

That's why the American Lung Association devoted four years of research and testing to develop the most effective ways—for the 1980's—to help smokers quit on their own. Or in a group.

COST OF SMOKING

Cigarette smoking is the major preventable cause of death and disability. In just one year alone—in 1982—340,000 Americans died as a result of cigarette smoking. Millions more lived on with crippled lungs and overstrained hearts.

The emotional, physical, and financial costs to smokers—and to their families—are incalculable.

But the burden afflicts many more than smokers, their families and friends – terrible though their toll is. It affects companies smokers work for, coworkers, the insurance industry and people who pay its premiums, the entire health care system in this country, and taxpayers.

Measured in financial terms alone, the effects of smoking cost our society more than $38 billion every year:

- Direct health care costs for smoking-related illnesses, much of which all taxpayers and everyone who has health insurance must assume, total an estimated $13 billion every year.
- Lost productivity and wages due to these illnesses account for an additional $25 billion yearly loss.

Of the hundreds of billions of dollars spent by Americans on health care each year, the insurance industry estimates that employers pay nearly half these costs.

Smoking is clearly far more than the smoker's private business. The burden is staggering.

CREATING A HEALTHIER WORK ENVIRONMENT

People are any employer's major asset. A healthy workforce is crucial.

Each year more than 80 million work days are lost due to smoking, and other employees must help pick up that workload. Smokers, on the average, have 35-45% higher absenteeism rates compared to nonsmokers.

Smokers also have twice the deaths due to coronary heart disease and ten times the risk of developing lung cancer.

Smoking is responsible for 85% of all lung cancer and more than 80% of such long-term severe lung disease as emphysema and chronic bronchitis.

Reports of distress from nonsmokers indicate that smoke-filled spaces interfere with their productivity

and morale. And there is the constant worry that passive smoking may have effects on their health.

Some nonsmokers have won disability, unemployment and worker's compensation awards because of exposure on the job. The pace is accelerating.

When employers indicate to their employees a concern about the effects of smoking—and help provide ways to help smokers quit and also protect nonsmokers from exposure—this in itself has enormous impact. And more and more companies have begun to take seriously their responsibility for providing a healthful and safe environment.

Although the precise cost to employers of an individual employee who smokes is hard to pinpoint, those estimates from national experts in economics range from $400 to $4600 every year. But this varies tremendously from person to person. Although costs are of bottom line concern to any business, the health of its employees is primary for ongoing effectiveness. The loss of even one key person—from a heart attack resulting from cigarette smoking, from lung cancer, or emphysema—has galvanized many companies to establish worksite programs to help smokers quit.

COMPANIES WHO CARE

Although it's important to find out how successful antismoking programs are in any particular setting, that is only one narrow focus. The broader focus is the permanent climate that management and unions create by taking steps toward a healthier workplace for everyone. Companies and organizations can convey a "We care about you" attitude that is central to change.

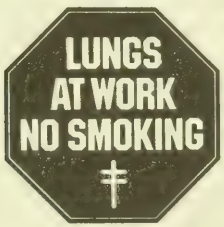
When no-smoking is seen as the desirable norm and smoking is viewed as destructive behavior that requires special assistance to stop, attitudes toward smoking are altered dramatically.

Permanent changes in smoking behavior do not happen overnight. Infrequent or one-shot interventions to stop smoking are not effective for long. Ongoing, long-term attempts and encouragement are important. For this, the workplace is the perfect setting.

- Employees spend substantial amounts of time at the workplace, sometimes as much or more time than they do at home.
- Programs to help smokers quit tend to take on special importance when they are offered as an employee benefit.
- Convenience is a special attraction.
- Coworkers and friends on the job can become strong, ongoing support groups to help smokers through the occasional tough times of quitting.

HIGH-RISK INDUSTRIES

Smoking in certain occupations greatly increases the threats to lung health because of the synergistic effects of tobacco smoke and other airborne hazards. Controlling smoking at the workplace – as well as exposure to other airborne hazards – is vitally important in these industries. *Only* supporting quit-smoking programs, the American Lung Association maintains, is insufficient action for employers in high-risk industries. In these cases, smoking programs should be an integral part of a comprehensive occupational health education and control program.



LUNG ASSOCIATION PROGRAMS

What Lung Associations offer companies and unions are programs that can be tailored to the particular needs of that company. How to involve employees—smokers and nonsmokers—depends on the working styles of the company, its internal publications, management approaches, union situation. Lung Associations can provide consultation to develop an individualized, ongoing approach.

In contrast to many new quit-smoking organizations that crop up overnight and disappear tomorrow, the American Lung Association has been in the forefront of the smoking issue for decades and has been fighting lung disease for most of the 20th century. Lung Associations plan to continue the fight as long as it is necessary.

Credibility, flexibility, and the possibility of a long-term relationship are the advantages of Lung Association programs at the workplace. For smokers, Lung Associations provide nationally-tested, top quality materials—available at minimal cost—with special emphasis on maintenance.

PROGRAMS FOR SMOKERS

Most people who quit smoking—more than 90%—do it on their own or with self-help materials. A much smaller number, often people who have smoked heavily and for long periods, need the special supports offered by group programs.

A) The FREEDOM FROM SMOKING self-help manuals are the most comprehensive quit-smoking guides ever developed by the American Lung Association. They are the result of four years of research and testing and an investment of a quarter of a million dollars. Many companies—such as IBM which distributed at corporate expense 30,000 sets of the manuals on request—say they are the best materials available anywhere. At most Lung Associations, the contribution suggested for the set of manuals is $10 or less.

The FREEDOM FROM SMOKING Manuals are the most comprehensive

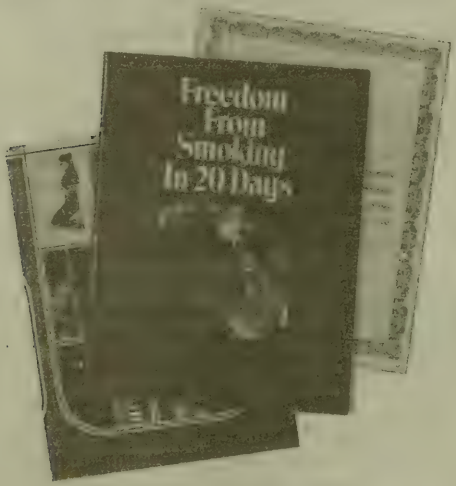
These manuals were developed by specialists...doctors, psychologists, people who truly understand your smoking problem. They know

MANUAL NO. 1

FREEDOM FROM SMOKING IN 20 DAYS

QUIT SMOKING Guides ever developed by The American Lung Association.

how hard it is to break this habit and they also know...from scientifically-gathered statistical data...that it can be done!

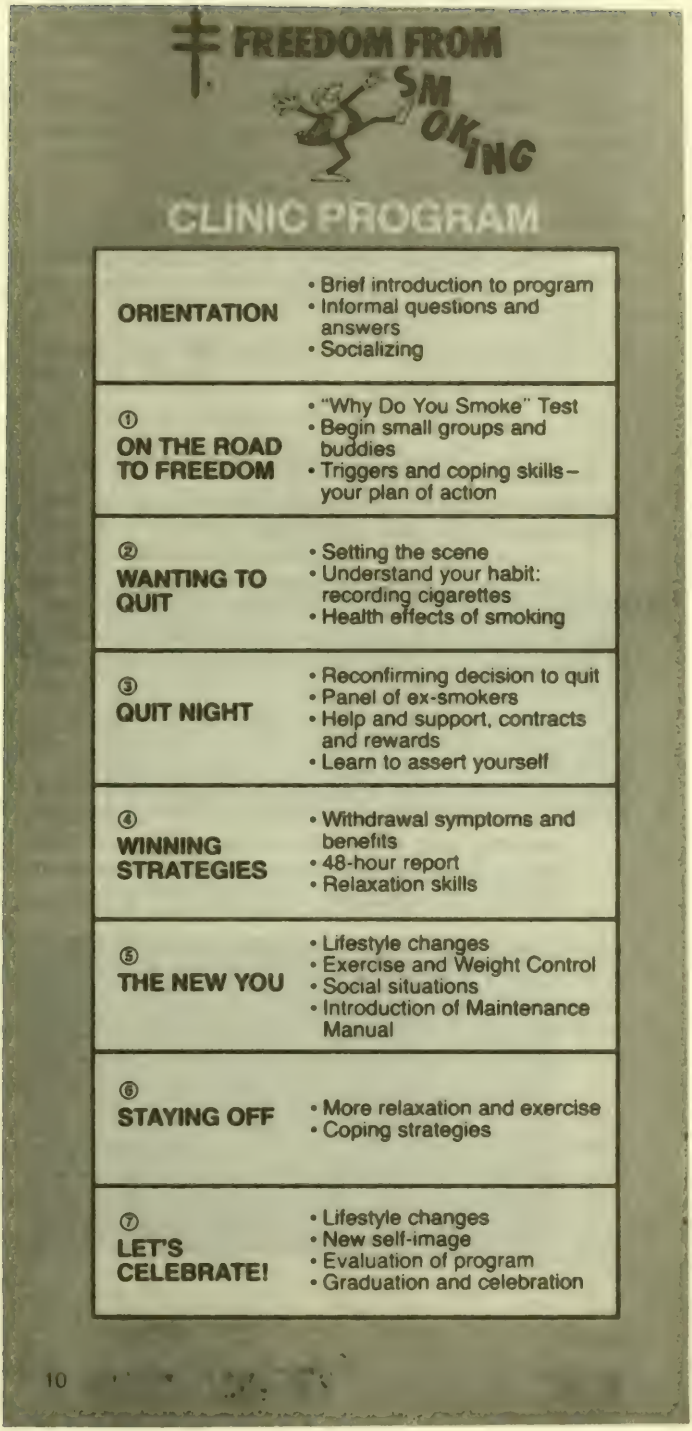


MANUAL NO. 2

A LIFETIME OF FREEDOM FROM SMOKING

also is fully-illustrated in 4 colors. It's a 28-page manual with extra materials added. This upbeat plan of action is designed to give you the help you need to stay off cigarettes once you've quit. And this time forever!

9


FREEDOM FROM SMOKING
CLINIC PROGRAM
ORIENTATION
• Brief introduction to program
• Informal questions and answers
• Socializing
① ON THE ROAD TO FREEDOM
• "Why Do You Smoke" Test
• Begin small groups and buddies
• Triggers and coping skills – your plan of action
② WANTING TO QUIT
• Setting the scene
• Understand your habit: recording cigarettes
• Health effects of smoking
③ QUIT NIGHT
• Reconfirming decision to quit
• Panel of ex-smokers
• Help and support, contracts and rewards
• Learn to assert yourself
④ WINNING STRATEGIES
• Withdrawal symptoms and benefits
• 48-hour report
• Relaxation skills
⑤ THE NEW YOU
• Lifestyle changes
• Exercise and Weight Control
• Social situations
• Introduction of Maintenance Manual
⑥ STAYING OFF
• More relaxation and exercise
• Coping strategies
⑦ LET'S CELEBRATE!
• Lifestyle changes
• New self-image
• Evaluation of program
• Graduation and celebration
10

B) Like the self-help manuals, the FREEDOM FROM SMOKING Clinics have been nationally tested. Test results showed the most successful format to be seven sessions conducted over a seven-week period. Smokers quit at the third session; the last four sessions emphasize maintenance of a new nonsmoking lifestyle. For people who want the support of a group, the clinics have special appeal. The staff of most Lung Associations have attended national workshops and are trained to conduct the clinics and also to train others—such as employee volunteers—to conduct the clinics.

A minimal fee—costs vary depending on location—is requested.

NONSMOKERS NEED PROTECTION

Few smokers are aware of the effects of their smoking on the entire society. But more and more of them are aware of the distressed reactions their smoking creates from those near-at-hand. This is particularly obvious at the workplace, where nonsmoking employees have expressed their concern to their smoking coworkers, personnel, unions, and management.

Two-thirds of the American workforce are nonsmokers.

In a recent study of 10,000 nonsmoking office workers, more than 50% of them reported difficulty working near a smoker; another 36% said they were forced to move away often from their desks or work stations because of second-hand smoke.

In a 1983 survey by the Gallup Organization, 75% (64% of smokers and 84% of nonsmokers) felt smoking was probably hazardous to the health of nonsmokers. And 69% (including 55% of smokers interviewed) said smokers should refrain from smoking in the presence of nonsmokers. Just as many smokers (64%) as nonsmokers preferred setting up separate areas for smoking at work.

What alarms nonsmokers, who are two-thirds of the workforce, are studies that indicate actual physiological effects of smoke on nonsmokers. Carbon monoxide levels in the nonsmokers' blood—as well as their blood pressure and heart beat—can rise after only thirty minutes in a smoke-filled room. This poses a dangerous, extra burden for those who already suffer from lung and heart diseases. And for people suffering from asthma, smoke may actually trigger asthma episodes.

Eye and throat irritations, headaches, and cough are reported by a high percentage of nonsmokers in many surveys. And a study of nonsmokers exposed to smoke at work for many years showed a dysfunction in the small airways of their lungs.

Some studies have found an increased risk of lung cancer in nonsmoking wives married to men who smoke. Although the studies are too few as yet to conclude a definite association between secondhand smoke and lung cancer, the findings have raised concern.

Since there are cancer-causing agents in cigarette smoke, it is not unreasonable to expect that inhaling these agents firsthand or secondhand could cause disease. Exposure to tobacco smoke, says the American Lung Association, may be similar to exposure to radiation: there are no safe levels.

SETTING UP A COMPANY POLICY OR GUIDELINE ABOUT SMOKING

Attitudes about smoking have undergone dramatic changes in the last few years. The overwhelming majority of nonsmokers say they do not want to be exposed to cigarette smoke. And a decided majority of smokers see their own smoking in negative terms.

Most states and localities have passed laws limiting smoking in a wide variety of public places. Some—an increasing number—also require that workplace smoking be controlled. A growing number of new laws and ordinances require that employers have written policies about smoking but do

not specify what these policies may be, only that management address the issue and make concerted efforts to protect nonsmokers.

> Employees are now looking to management for such policies. The divisiveness between smokers and nonsmokers is obvious. Adopting policies can remedy the situation and alleviate distress and personal clashes.

Adopting policies presents a bewildering prospect to some employers because they do not know how to begin and they fear disrupting work arrangements and alienating smokers. Instead, companies with policies about smoking report increased cooperation between employees and overwhelming acceptance of such policies.

A letter from the President or Chief Executive Officer, announcing that Lung Association stop-smoking programs will be made available to smokers—and that a policy to protect nonsmokers will be enacted by a certain date—is one way to begin.

Lung Associations have found that top management support is absolutely essential for the success of programs and policies about smoking.

Pace-setting, new policies being established by companies of all sizes now call for the smoker to respect the needs and requirements of the nonsmoker. In some companies, managers are asked to separate smokers and nonsmokers where this is requested and if it is practical. After providing stop-smoking programs for smokers, some companies have banned smoking altogether in work areas.

Major corporations now emphasize the rights of the nonsmoker and urge managers to find a reasonable accommodation when nonsmokers and smokers are in conflict. When such an accommodation is not possible, the corporations maintain that the rights of the nonsmokers should take precedence.

This completely reverses the usual situation in companies where no policies exist. There nonsmokers who attempt to protect themselves are too often viewed as disruptive and troublesome. A policy can change this perspective; the company then is clearly in support of the nonsmoker, who is pursuing a healthier lifestyle that benefits everyone, including the company.

Lung Associations have model policies from such companies as Control Data Corporation, American Telephone and Telegraph, McGraw-Hill, Pratt and Whitney Aircraft, and Rodale Press. Lung Associations can work with companies to create the kind of policy that best fits that particular worksite.

Ideally, stop-smoking programs are only one aspect – an outgrowth or a forerunner – of overall policies that indicate the company cares about the health and comfort of all its employees.

Policies are proof positive of that concern.

CONTACT YOUR LOCAL AMERICAN LUNG ASSOCIATION TO GET STARTED TOWARD A HEALTHIER ENVIRONMENT AT YOUR WORKPLACE.

AMERICAN
LUNG
ASSOCIATION®
The Christmas Seal People®

Should Tobacco Advertising And Promotion Be Banned?

SHOULD TOBACCO ADVERTISING AND PROMOTION BE BANNED?

The American Lung Association has long supported action to end advertising and promotion of cigarettes and other tobacco products and is now campaigning in favor of federal legislation for a comprehensive advertising and promotion ban.

Because of the grave hazards posed by tobacco products, Congress passed legislation barring cigarette advertising on radio and television in 1971. But the tobacco industry now spends billions of dollars on other forms of advertising and promotion, much of it directed to young audiences.

The U.S. Surgeon General has announced the goal of a smoke-free society by the year 2000. Yet today, as we near that goal, ninety percent of all smokers are starting the habit by age 19, and sixty percent begin by age 16. Obviously, a new generation of young people—our children—are still being recruited by the tobacco industry's advertising and promotion campaigns. We must not let that continue.

According to the U.S. Surgeon General, smoking is "the chief single avoidable cause of death in our society and the most important public health issue of our time." Here are the facts:

- It is estimated that 434,000 Americans die prematurely each year from diseases caused by smoking (U.S. Public Health Service), accounting annually for 20 percent of all deaths in the U.S.

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- The 1988 report by the U.S. Surgeon General on smoking warns that nicotine in tobacco products is as addictive as heroin and cocaine. According to the U.S. Public Health Service, nicotine addiction is "the most widespread example of drug dependence in our country."
- More than 50,000 medical and scientific studies have demonstrated beyond doubt that smoking is a cause of fatal disease.
- The total number of people who die from diseases caused by smoking each year is larger than the combined total of deaths from alcohol, illegal drugs, auto and all other accidents, and all homicides and suicides in the U.S.
- Millions more Americans suffer disabilities from diseases caused by smoking, including lung cancer, emphysema, and heart disease. Smoking-related diseases cost our nation up to $65 billion in lost productivity and health expenditures annually, according to the U.S. Office of Technology Assessment.
- The 1986 report by the U.S. Surgeon General entitled "The Health Consequences of Involuntary Smoking" concludes that involuntary smoking is a cause of disease, including lung cancer, in healthy nonsmokers.

The questions below present issues raised by the campaign to ban tobacco advertising and promotion and the position of the American Lung Association on these issues.

HOW IMPORTANT IS CIGARETTE ADVERTISING AND PROMOTION?

Cigarettes are the most heavily marketed consumer product in America, even though they cannot be advertised on television or radio.

Tobacco companies currently spend more than $3 billion a year ($3.3 billion in 1988) to promote and advertise their products, and have increased their spending more than nine times since 1971 when advertising on radio and television was banned.

The tobacco industry is the largest advertiser in the print media, including magazines, newspapers, and also outdoor billboards.

WHAT IS THE INFLUENCE OF TOBACCO ADVERTISING AND PROMOTION ON CHILDREN AND TEENAGERS?

Tobacco companies say that their marketing is not aimed at children and teenagers, but much of it reaches and attracts young people.

Experts believe that children are extremely receptive to advertising images. Even before they can read, children see the slick, youthful images of cigarette smokers on billboards and posters.

Teenagers see smoking ads in sports, news, women's and entertainment magazines and in newspapers. Tobacco companies sponsor major sports and entertainment events that attract young people.

Tobacco advertising and promotion project images of smoking as fun, sexy, glamorous, macho, and—most insidiously—healthful. Other images link smoking with success

and "the good life." A former chief executive of R.J. Reynolds, Inc. said that the ads associate smoking "with happy occasions," and "to a degree with love and the outdoors."

WHAT IS THE WIDER IMPACT OF TOBACCO ADVERTISING AND PROMOTION?

The fact that cigarette ads are allowed to appear in print and elsewhere, even with health warnings, indicates to many—especially young people—that tobacco products "can't be that bad" or the government wouldn't let them be advertised.

The Federal Trade Commission has noted that "some ads use language which directly contradicts the required health warnings and scientific evidence that smoking is dangerous to health and perhaps life itself."

Pervasive tobacco advertising and promotion adds to the difficulties of those trying to stop smoking by projecting the impression that smoking is a normal activity indulged in by almost everyone. This is not true: less than 28.8 percent of American adults are smokers. While this is still a tragically large figure, it hardly constitutes a majority of the population.

Another strategy of tobacco advertisers is to mount aggressive campaigns aimed at specific segments of the population, such as women, Blacks and Hispanics, and blue-collar workers. Various cigarette brands, for example, are targeted directly at women, and eight of the twenty magazines receiving the most cigarette advertising revenue in 1985 were women's magazines.

Other magazines and newspapers that carry tobacco advertising also seem to be influenced by having these ads on their pages: by and large, they run fewer articles on the dangers of smoking. One recent study of 12 major U.S. magazines carrying cigarette advertising showed that over 12 years, eight of the magazines published *no articles* on the hazards of smoking, despite regular articles on other health issues. The American Public Health Association says that "magazines accepting tobacco ads are addicted to the revenues generated by tobacco advertisements."

THE TOBACCO INDUSTRY CLAIMS ITS ADVERTISING IS DESIGNED TO PERSUADE SMOKERS TO SWITCH BRANDS, NOT ATTRACT NEW SMOKERS. IS THIS ACCURATE?

The founder of a major advertising agency, Emerson Foote, has commented on this claim: "The industry knows it's nonsense. I am always amused by the suggestion that advertising, a function that has been shown to increase consumption of every other product, somehow fails to work with tobacco products."

With only 10 percent of smokers switching brands each year, the industry could not recover the more than $3 billion it spends annually by directing its advertising and promotion only to this group.

Because of deaths from smoking and other causes, and because of people who quit smoking, the tobacco industry must recruit two million new or former smokers a year—or increase consumption among remaining

smokers—just to maintain sales at present levels. Advertising and promotion help to accomplish this.

HOW IMPORTANT ARE PROMOTIONAL ACTIVITIES BY TOBACCO COMPANIES?

Spending for promotion by tobacco companies now accounts for more than 68% of all marketing, reaching $2.2 billion in 1988, and has surpassed advertising spending. Promotion includes free sampling of the product, allowances paid to retailers for shelf space and positioning products, distribution of promotional products imprinted with cigarette brand names, endorsements and testimonials, and sponsorship of sports, musical and other public entertainment and cultural events.

Many promotional displays do not include the Surgeon General's health warnings required on print advertisements. Brand names used in sponsoring events—such as those that appear on billboards at baseball games—are seen by audiences of millions of people and are often broadcast on television, undercutting the advertising ban.

Sponsorship conveys an image of corporate "good citizenship" and generosity, and connects tobacco products, which are toxic and addictive, with exciting—and healthy—sports events and other popular activities. Unfortunately, a number of under-funded cultural organizations eagerly seek funds from tobacco companies and become dependent on them.

WHAT IS THE IMPACT OF THE DISTRIBUTION OF FREE SAMPLES?

The distribution of free cigarette samples is a particularly insidious form of promotion that has increased significantly. Spending by tobacco companies for distribution of free samples rose from $12 million in 1970 to $74.5 million in 1988.

Although there are laws banning distribution of samples to minors, they are often handed out at youth-oriented activities, such as sports events and rock concerts. There is evidence that many children receive free samples. And smokers trying to quit are very vulnerable to these offers.

THE FIRST AMENDMENT GUARANTEES FREEDOM OF SPEECH AND OF THE PRESS. DOESN'T THAT APPLY TO TOBACCO ADVERTISING AND PROMOTION?

The First Amendment fully protects political, religious and cultural speech. But advertising and promotion are commercial speech, carried out for economic gain, and may be regulated. Cigarette advertising has been barred from radio and television, and this has been sustained by the Supreme Court.

The Supreme Court has set out criteria relevant to a comprehensive ban on tobacco advertising and promotion in two recent decisions:

In the first, it indicated that commercial speech which is misleading may be prohibited, and also that commercial speech may be banned where there is a substantial govern-

ment interest at stake which the ban would advance.

Advertising and promotion of tobacco products *are* misleading and deceptive. They project images of enjoyment and satisfaction that obscure the addiction and fatal diseases linked to tobacco, and that contradict health warnings also carried by the products themselves. The substantial government interest in a ban is clear, given the record of addiction, disability and death caused by tobacco products.

In the second case, the Supreme Court indicated that since the government can restrict and prohibit products or activities deemed harmful (such as cigarettes, alcoholic beverages, or gambling) then the government also has the authority to restrict or bar advertising for these products or activities.

DOES THIS MEAN THAT EVEN THOUGH IT IS LEGAL TO SELL TOBACCO PRODUCTS, ADVERTISING AND PROMOTION FOR THEM CAN BE BANNED?

Yes. Many products, such as pesticides, saccharin, prescription drugs, and firearms, do carry sales or marketing restrictions. Stocks and bonds cannot be advertised directly.

Cigarette sales to minors are barred in most states, and television and radio advertising for them have been banned. The cases cited above indicate that a comprehensive ban can be imposed.

WOULD A BAN ON ADVERTISING AND PROMOTION DENY PEOPLE "FREEDOM OF CHOICE" ABOUT SMOKING?

This is a claim often made by the tobacco industry, but the mass marketing of products that are highly addictive and the leading cause of fatal disease hardly advances Americans' freedom of choice.

Children do not have the knowledge and experience needed to make informed choices and are the segment of the population most vulnerable to tobacco advertising and promotion.

One survey found that more than half of high school seniors who smoke have tried unsuccessfully to quit, and while only five percent believed they would still be smoking years after graduation, in fact 73 percent were still smoking eight years later. A large majority of adult smokers say they would like to quit, but most who try are unsuccessful. Their freedom of choice—the choice to be free of tobacco—has been severely limited.

WHY PICK ONLY ON TOBACCO WHEN SO MANY PRODUCTS SEEM TO CAUSE CANCER?

Tobacco is uniquely harmful and not comparable to any other consumer product. Tobacco is the only legal product that causes death and disability *when used as intended.*

Yet mainly because of the influence of the tobacco industry, tobacco products have been exempt from the laws and regulations passed by Congress to protect our health and safety—including the Food, Drug and Cosmetic Act; the Toxic Substances Act; and the

Consumer Products Safety Act—and from control by the agencies enforcing these laws.

The National Council on Drug Abuse has stated in support of a complete ban on tobacco advertising and promotion: "To permit the current massive promotional activities for this product is, in our opinion, both highly immoral and inconsistent with our handling of nearly all other products in the society."

IF TOBACCO PRODUCTS ARE SUCH A THREAT, WHY DOES THE GOVERNMENT ALLOW THEM TO BE SOLD?

If cigarettes and other tobacco products were being newly introduced, it is not likely that they would be approved for sale because they are so toxic and addictive.

Tobacco products have been mass produced and marketed for decades and it has taken many years for their deadly effects to be scientifically documented—evidence still disputed by the tobacco industry. Now, millions of Americans are dependent on or addicted to tobacco and a prohibition on sales is not feasible.

However, a ban on tobacco advertising and promotion can contribute to ending the epidemic of tobacco-caused addiction and disease that mass marketing of these products has helped to foster.

IS TOBACCO ADVERTISING AND PROMOTION TAX DEDUCTIBLE?

Yes. Under current tax regulations, tobacco companies are allowed to deduct the costs of advertising and promotion from their

taxes as a business expense. This saves them in excess of $1 billion a year in taxes, money that otherwise would go to the U.S. Treasury.

CAN A BAN ON TOBACCO ADVERTISING AND PROMOTION BE SUCCESSFUL?

There are more than 20 countries that have total or near-total advertising bans on cigarettes, including Finland, France, Iceland, Italy, Norway, Belgium, Singapore, Sweden, and Canada.

The bans have been effective in Finland, Norway, and Sweden, where the governments have combined them with vigorous educational campaigns on the dangers of smoking. There has been a significant decline in smoking by teenagers in these countries.

WHAT WOULD BE INCLUDED IN A COMPREHENSIVE BAN ON TOBACCO ADVERTISING AND PROMOTION?

Proposed legislation for a comprehensive ban would bar: radio and television commercials (already prohibited since 1971 for cigarettes and since 1986 for smokeless tobacco); newspaper and magazine advertisements; billboards, posters, decals, and other promotional materials; and free samples, coupons and premiums.

Also banned would be sponsorship of athletic, cultural, or other events bearing the brand name or logo of tobacco products, and marketing other products or services bearing the brand name or logo of tobacco products.

IS THERE PUBLIC SUPPORT FOR A COMPREHENSIVE ADVERTISING AND PROMOTION BAN?

In an 1987 survey of public attitudes conducted by the American Medical Association, a majority indicated their support for a ban on cigarette advertising. In the poll, when people were asked if they would favor or oppose a ban on advertising tobacco products,—55% (61% nonsmokers and 42% smokers)—said that they favored such a ban.

But powerful lobbying forces representing the tobacco industry, as well as advertising agencies and some in the print media, continue to be relentless in their opposition to any restriction on tobacco advertising or promotion.

WHAT IS BEING DONE IN THE U.S. TO RESTRICT AND BAN TOBACCO ADVERTISING AND PROMOTION?

Legislation to ban all tobacco advertising and promotion has been introduced in Congress. Other legislation has been proposed that would end tax deductions by tobacco companies for spending on advertising and promotion and give the government authority to regulate misleading advertising related to tobacco products.

The American Lung Association has joined with the American Cancer Society and the American Heart Association to form the Coalition on Smoking OR Health to support these legislative initiatives.

Because of the influence of the tobacco companies, a provision of the 1969 federal law providing for health warning labels on ciga-

rettes also prohibits states and cities from restricting tobacco advertising and promotion. The American Lung Association is supporting efforts to revoke this provision.

States and localities have been able to take other steps, such as barring tobacco advertising on state property, in public transit vehicles, and in publicly-owned stadiums and other facilities.

More than 30 national magazines no longer accept cigarette advertisements and have been given special awards by the American Lung Association for their policies.

WHAT CAN I DO TO SUPPORT THE CAMPAIGN?

Contact your local American Lung Association (listed in the white pages of your telephone directory) to find out about activities in your area. You can work with your Lung Association to build support for federal, state and local measures. You can also:

- Write to publishers of magazines and newspapers to oppose tobacco advertising and promotion and encourage articles on the harmful effects of smoking.
- Help children and teenagers to see beyond slick tobacco ads to understand the dangers of smoking and the goal of a smoke-free society.
- Encourage smokers trying to quit.
- Ask your local American Lung Association for these materials:

- Freedom From Smoking® For You and Your Family
- Help a Friend Stop Smoking
- TEAM UP FOR FREEDOM FROM SMOKING® at Work

- Freedom From Smoking® For You and Your Baby
- Facts About Cigarette Smoking
- Facts About Nicotine Addiction and Cigarettes
- Facts About Secondhand Smoke

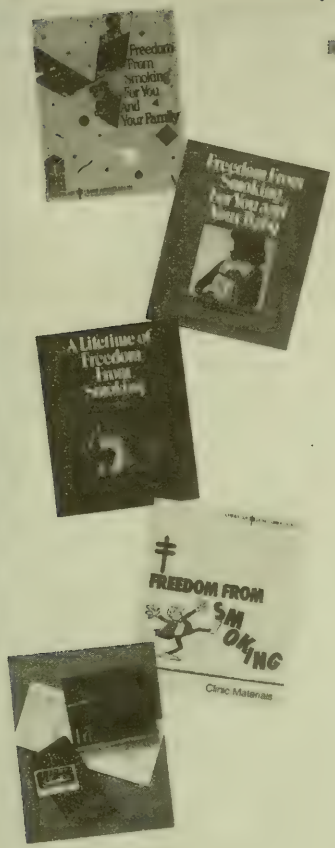
WHAT OTHER PROGRAMS IS THE AMERICAN LUNG ASSOCIATION CARRYING OUT TO PROMOTE A SMOKE-FREE SOCIETY?

The American Lung Association has extensive programs aimed at encouraging young people not to start smoking, helping smokers to stop, and limiting the marketing of tobacco products.

ALA supports federal legislation to increase the federal excise tax on cigarettes, which is one of the most effective incentives for discouraging Americans from smoking. In addition, ALA believes that revenues from a cigarette excise tax increase should be used for public education programs on the hazards of smoking.

ALA also supports the adoption of new federal regulations to give the Food and Drug Administration the authority to regulate tobacco advertising, access to tobacco products, and labeling of tobacco products. Another federal measure supported successfully by ALA was a smoking ban on domestic airlines (1990).

The health hazards of smoking affect all of us—smokers and nonsmokers alike. Your local American Lung Association can provide information about these issues and assistance in dealing with them. Get in touch with your local ALA today.



FREEDOM FROM SMOKING®

Now is the time to quit smoking! This minute, today, this week, before your next birthday! Taking it a step at a time is what the American Lung Association's Freedom From Smoking® Programs are all about. One of our broad range of programs is ideally suited to helping you make this your last attempt at quitting smoking, forever.

AMERICAN LUNG ASSOCIATION®
The Christmas Seal People®

AMERICAN LUNG ASSOCIATION®
The Christmas Seal People®

0399 5/90

1

Freedom From Smoking® For You and Your Family

A step-by-step guide to help you quit smoking so that you and the people around you can enjoy a better life. The easy-to-read, 56-page guide is planned so you can use any step that meets your own special needs during the quitting process. It covers important stop-smoking issues such as weight gain, addiction, and coping strategies. The manual includes a quitting calendar and easy-to-use records to help you get to know your own smoking habit. Specific techniques help you avoid sliding back once you've quit and help you stay on the road to a smoke-free lifestyle, forever.

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2

Freedom From Smoking® For You and Your Baby

A ten-day self-help program prepared by two health care experts: The American Lung Association and The Harvard Community Health Plan. The program recognizes that pregnancy is a time of increased stress, emotional and physical changes, and new demands on your time. This step-by-step smoking program includes a 32-page full-color manual, a poster-size progress calendar, a special exercise chart for expectant mothers, and a pocket reminder. Also available is an audiocassette featuring special aerobics and relaxation techniques for pregnant women.

Make a decision today to quit smoking! Your American Lung Association will help you every step of the way.

3

A Lifetime of Freedom From Smoking®

Maps out strategies to help the new nonsmoker stay that way for a lifetime. This 28-page maintenance manual gives suggestions on how to stay quit and deal with issues such as controlling weight and coping with social situations as a nonsmoker. Staying quit is the hardest part to quitting, and this manual helps you develop a nonsmoking habit that will last a lifetime.

4

In Control® A Video Freedom From Smoking® Program

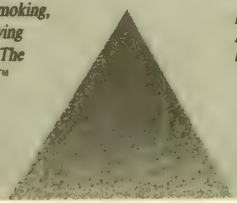
Based on the most recent smoking and nicotine research available. You watch one 9-minute video segment every day for 13 days. Each segment gives you motivation, encouragement, and specific techniques on how to become a permanent nonsmoker. ***In Control®*** *includes a 2-hour video cassette (105 minutes total viewing), a 124-page viewer's guide, and an audiocassette featuring relaxation techniques. The American Lung Association can also provide* ***In Control®*** *users with special guides for maximizing group settings at work or in the physician's office.*

5

Freedom From Smoking® Clinic

An eight-session group program led by experts who understand your motivations and your rationalizations about continuing to smoke. The program uses a positive behavior change approach that teaches you how to become a nonsmoker. Special attention is given to developing a quitting strategy, dealing with recovery symptoms, controlling weight, managing stress through relaxation, to assertiveness techniques, and to staying off cigarettes. For many smokers — especially those who have tried to quit before — group support can make the difference in helping to stay smoke-free. Contact your Lung Association for price and schedule information.

Time's passing and now is the moment to begin living a healthier, happier, smoke-free lifestyle. Gain freedom from smoking, and you'll join a growing group of Americans: The Smoke-Free Family! ™



Order Form

Make the first step towards gaining your Freedom From Smoking® and join the Smoke-Free Family! ™ *Just fill out this order form and mail it today.*

Name _____
(Please Print)

Address _____

City _____

State _____ Zip Code _____

Item	Unit Price	Quantity Ordered	Total Price
Freedom From Smoking® for You and Your Family	$7.50		
Freedom From Smoking® for You and Your Baby Self-Help Manual	$8.75		
Freedom From Smoking® for You and Your Baby Audiocassette	$6.00		
In Control®: A Video Freedom From Smoking® Program	$59.95		
A Lifetime of Freedom From Smoking®	$6.75		
	Total		

☐ *Send me schedule and price information about your Freedom From Smoking® Clinics.*
When ordering multiple copies of a single item, check with your local Lung Association for possible discounts and savings.

☐ *Payment Enclosed* ☐ *Bill me*
Allow 2-4 weeks for delivery.

Mail this order form to your local American Lung Association. You can find the address and telephone number in the white pages of your telephone book.

Update Details Vol. XIV No. 39 - October 18, 1991

Quit Rates for Freedom From Smoking® Materials

To respond to public and media inquiries*, ALA is using the following quit rates for Freedom From Smoking® programs. Generally, when quoting rates the **nonsmoking prevalence** is given which is the percentage of smokers who at a 12 month follow up have not smoked in the past 30 days.

•Freedom From Smoking® Clinic 28.6%

For more information, please refer to December 11, 1987 Update/DETAILS.

•Freedom From Smoking® - In Control Video 21%

For more information, please refer to the American Journal of Public Health, 1988, Vol. 78, No. 9.

•Freedom From Smoking® In 20 Days and A Lifetime of Freedom From Smoking® 18%

For more information, please refer to the American Journal of Public Health 1984, Vol. 74, No. 11.

•Freedom From Smoking® For You And Your Family 11-21%

Nonsmoking prevalence is defined in this study as the percentage of smokers who at a 6 month follow up have not smoked in the past week. For more information, please refer to Update/DETAILS, August 10, 1990.

•Freedom From Smoking® For You and Your Baby 29% and 35.6%

29% of smokers using FFS/YYB were not smoking at eight weeks after the baby's birth. 35.6% of smokers using FFS/YYB plus clinician involvement and follow up materials were not smoking eight weeks after the baby's birth.

While the nonsmoking prevalence rates seem low, it is important to point out that today's smokers represent a segment of the population who have been heavily smoking for many years and subsequently many of these smokers are addicted to nicotine. These percentages reflect the need to view today's smokers in the different stages of the smoking behavior change process:

1. Pre contemplation (not thinking about quitting)
2. Contemplation (thinking about quitting)
3. Action (ready to make or making a quit attempt)
4. Maintenance (staying quit)
5. Relapse

In this way we can determine how best to meet their needs and to move smokers from thinking about quitting to making a quit attempt or to try to quit even after several relapses. Our measure of success should not just be quit rates but whether we move the smoker into a different stage in the quitting process.

For additional information on Freedom From Smoking® Quit Rates please contact Jeanne Nametz, Associate, Smoking or Health at (212) 315-8723.

* Please inform your Freedom From Smoking® facilitators that we are no longer collecting data for the ALA FFS® Intake and End of Clinic Questionnaire in 1986.

April 23, 1993

SMOKE-FREE CLASS OF 2000

1993 STATUS REPORT

Education Component
Development, Production and Dissemination of Materials

Fifth Grade Activity Kit: Over 135,000 kits were distributed during the 1992-93 school year, depleting the original supply. By using available posters and audio cassettes and ordering additional envelopes, teaching guides and pre/post tests as needed, we were able to collate an additional 2750 5th grade kits. There are currently 2,733 5th grade kits in inventory. However, several states have some extra kits should we need additional ones over the next couple of years.

Sixth Grade Activity Kit: The production of the 6th grade activity kit is on schedule. All of the mechanicals were delivered to the American Heart Association in Dallas on March 24. The materials were reviewed by a variety of people--staff and volunteers--at the local and national level.

The completed video which was delivered to the video reproduction vendor on April 13, is excellent! The production quality is very good and the cast is extremely professional. I was present for the filming of one segment of the video in New York. The producer, Myles Fox, pulled in a lot of favors and convinced many talented people to donate time and materials to the effort. Keshia Knight Pulliam is featured in the video and the smoke-free kids are played by are young actors enrolled in an acting school. I think we will get a lot of mileage out of this package.

Orders are in from almost all of the states. It looks as though the initial distribution will be approximately 50,000. We had 75,000 packages produced, plus all of the components will be available separately. There should be plenty available to fill orders for the next couple of years. The kits will be mailed to the field in June. The price will not exceed the $3.20 estimate and may be closer to $3.00 per package--a real bargain for a video, 3 teaching guides and 3 posters.

Seventh Grade: After a thorough review process of two excellent proposals, Sharon Jaycox, Greg Schneider, Beth Stevenson and Gail Joyce selected the vendor to develop the materials for grades 7 - 9. Education Development Center, Inc. was the final choice of the selection committee because of EDC's experience with developing junior high health education curriculum, their connections with other agencies in the health network and their school contacts.

While we had the option of choosing a different vendor for the design and layout of the materials, the decision was made to let EDC handle that aspect as well to keep the process as streamlined as possible. The production and distribution phase will continue to be coordinated by the American Heart Association production department.

The contract, which will be effective July 1, 1993, for the development of the content area is being reviewed by AHA's Legal Department. It will be ready for signature by the end of April. The video component will be covered by a separate contract which will be finalized as soon as possible.

I plan to meet with Chris Blaber, Project Manager from EDC and her team in June to review the action plan and production schedule.

Coalitions/Working Groups

We continue to send out an UPDATE every other month to the state coordinators, Field Advisory Group and national spokespersons. In addition, an abbreviated version is sent to each agency for distribution through their own systems.

Conference calls were conducted in January and February of this year. The purpose of the calls was to clarify the procedures for conducting the Bookcover Contest and workout any problems; to request each state to provide information from the Pre/Post test in the kit and to provide information about the 6th grade kit and how to order. These conference calls have become a valuable means of communicating to the field and sharing information with each other. Forty states participated in this last round. Another round will be conducted late this summer--after the kits have been mailed.

As is usual, some states have stronger coalitions than others and occasionally in a few states the program almost dies--usually because of changes in personnel or the structure of the participating organizations, or budget constraints; however, the problems seem to work themselves out as in the case of California. After losing their state coordinator, suffering a major budgeting setback, and threatening to jettison the program completely, the state is back on track stronger than ever. Each local coalition was required to provide funding for the cost of the materials--be self-supporting. They report that the response was better than expected and orders exceeded initial projections. Two other states are currently working through their problems. Maine and Mississippi.

This past year, I visited coalitions in Texas and Georgia and have been invited to attend the graduation ceremony in St. Louis, Missouri.

Communications

For information on national media activity, refer to the Communications Report.

The Video News Release or Information Video and B Roll proved to be very popular. Approximately 400 information videos and 50 B Roll have been ordered by the field. Many states used the video in teacher trainings and volunteer and staff orientation in addition to TV shows.

This year we established an 800# that was provided in releases to professional

publications--primarily for teachers to use to request information about the project and the contest. Since inception in September, we have received approximately 1500 calls, with the majority coming in the months when stories appeared in Weekly Reader, Instructors Magazine, NEA Today and Good Housekeeping (November, February, March and April). We responded to the calls with a letter, Question & Answer Sheet about SFC 2000, order forms for education materials and supplemental items, and a 5th grade kit if specifically requested. We included the name, address, and telephone number of the state coordinator and sent a copy of the letter to the state coordinator for follow up.

Because of the large volume of inquiries from parents as well as teachers, we plan to produce a general brochure about the project. A contact from [illegible] who teaches a college level advertising course wants to give the brochure to her students this fall as a special project.

Program Impact

Based on early returns of the Pre/Post Test, the fifth grade materials were very effective. In every classroom reporting, scores were significantly improved after participating in the project. We also received some letters and paragraphs from the students attesting to the impact of the material and the desire to be "tobacco-free."

We continue to receive Teacher FeedBack Sheets from the Middle School Activity Kit- all responses are very positive.

Lederle Laboratories and National Bookcover Contest

As of April 20, we had received entries from all but three states: Maine, Mississippi and Alaska. Many of the states planned special events surrounding the contest. In Vermont, Dr. C. Everett was one of the judges, in [illegible] states the governor recognized the top winners, and in others entries are on display at the capitol building or in some other prominent public place.

A box of prizes for the state winners and their classrooms will be mailed to each state the first week in March. Prizes included T shirts and certificates for all winners, sets of educational materials from ACS, AHA and ALA. Savings bonds are being processed now and we hope to have them to the winners by the first week in May.

Initial judging for the final four will be conducted by the SFC 2000 Steering Committee and Field Advisory Group and a group of 6th graders from Albuquerque. The top sixteen to twenty entries will be forwarded for final judging by Lederle representatives.

On March 15 a letter was sent to Hillary Rodham Clinton signed by the three CEOS's

requesting her participation in a recognition ceremony for the four finalists in Washington, D.C. in late May or early June. An AHA volunteer has been the driving force behind this proposal and he has contacts who are Friends of Bill and Hillary, who offered to give the request a personal touch. Unfortunately, the timing could not have been worse--the letter arrived during the "STORM" and the week her father had his stroke. As of April 29, we have not heard anything, but Dr. Bill Tansy is continuing to track the progress of the request. In the beginning it looked very promising--but now with the death of Mrs. Clinton's father and the delay of the announcement of the Health Care Plan, which I understand will occur sometime between May 25 and 28, the possibility of her participation appears increasingly remote.

Budget and Funding

A complete budget report has been provided to Sharon Jaycox, Chair.

Funding proposals have been presented to Lederle Laboratories and Mutual of America by AHA Corporate Relations on behalf of the coalition. As of this date, there has not been a definite answer from either corporation. We are told that Lederle "loves" this project and it is possible that there will be some level of funding for next year. Mutual of America expressed an interest in funding a coalition project.

1992
STATUS REPORT

SMOKE-FREE CLASS OF 2000

Background

In April of 1992 Richard P. Grimes, Deputy Managing Director, National Programs, American Lung Association presented to the CEO's of ACS, AHA, and ALA a status report on the Smoke-Free Class of 2000. (See Attachment A) That report summarized progress made between 1988 and 1992. This document reports on the progress made since that time.

Accomplishments

1. Outreach

For school year 1992-1993 all 50 states plus Washington, D.C., Puerto Rico, the Virgin Islands and Dependent Schools Overseas ordered over 132,000 fifth grade Smoke-Free Class of 2000 (SFC2000) Activity Kits for distribution to teachers throughout the year. Each packet contained a teacher's guide with a mock TV game show script, a take home activity, many follow-up activities, a list of school health education materials available from ACS, AHA, and ALA; an audio cassette; two posters; and a pre/post test for students. The price of the kits was $.80. Also available were a variety of supplemental items including T-shirts, book marks, pencils, stickers, etc.

2. Fundraising

Through the leadership of the American Heart Association's Corporate Relations Department, a corporate sponsor was secured for SFC2000. Lederle Laboratories has committed approximately $375,000 to partially support the program for a period of 12 months. This grant helps to offset the cost of developing and printing the 5th and 6th grade materials and has provided an exciting focus to the 1992-93 school year through the incorporation of a nationwide bookcover design contest.

3. Coalition Activity

At the state and local levels coalitions make SFC2000 a reality. A study of the 50 plus local SFC2000 locations, soon to be published in Health Education Quarterly, the premier journal in the public health community, found that 95% of all SFC2000 coalitions rated themselves moderately or very active. Each SFC-2000 coalition includes on average at least five organizations. In addition to the three founding agencies (ALA, ACS, AHA) state education, health department and medical society groups are part of the coalition.

4. Volunteer Participation

Volunteers and staff from the three agencies, working in state and local level coalitions, distributed over 130,000 4th grade activity kits and 132,000 5th grade kits to elementary schools nationwide. Coalitions not only use volunteers to distribute materials to schools, and conduct teacher training, but also to provide administrative support, to conduct follow-up activities in schools, for marketing, publicity, fundraising and organizing special events. SFC2000 volunteers are recruited from those already involved with existing agencies, physicians, public health nurses, school nurses, retired teachers, school board members and parents.

5. Impact at the Field Level

Great things are happening in the field. Not only is the educational message about making the healthy choice to avoid tobacco use getting out there, but also children are becoming advocates for themselves and for their communities. Further to our collective benefits is that these new advocates are our future volunteers. Below is just a sampling of activities in a few states.

North Dakota:

Two young members of the Smoke-Free Class of 2000 from Marmarth, North Dakota went to their school board to ask that their school be made tobacco-free. Citing information learned from the SFC2000 classroom materials, the boys convinced the school board to vote YES. The boys put up no smoking signs throughout the school and placed cigarette butt cans at the entrance to the school to encourage adherence to the new smoke free policy for which they advocated.

Massachusetts:

In Somerville, a densely populated, blue collar city, SFC2000 kids participated in the bicentennial parade. Dozens of kids in their SFC2000 t-shirts rode in a float with signs from each of the three agencies and one that said "Thank You Somerville for Giving Us a Smoke-Free Future" acknowledging recent restrictions on tobacco use in schools.

On October 6, the SFC2000 coalition and New England Cable News presented the first 30 minute cable in the classroom teleconference focusing on tobacco use prevention. Keshia Knight Pulliam the SFC2000 spokesperson was the featured guest. The timing was perfect as this "Cable in the Classroom" program reached every 5th grade classroom in the state and was linked to the upcoming referendum on increasing the cigarette excise tax in Massachusetts. (Question I, the referendum to increase the excise tax on cigarettes in Massachusetts passed on November 3, 1992)

Texas:

A conference held this fall entitled "Tobacco-Free Texas: Building a Partnership for a Healthy Texas" brought together key health care, school worksite and community organizations to discuss how best to deal with tobacco issues in the state. One of the sessions focused on the Smoke-Free Class of 2000 as an effective strategy to link classroom education with public policy. The keynote speaker for the conference strengthened this link by endorsing the SFC2000 as a means to linking comprehensive school health education, tobacco free schools and students as advocates for a Smoke-Free Society.

6. Media Coverage and Public Awareness

Media coverage of the Smoke-Free Class of 2000 has continued to be steady and, in cases where Keshia Pulliam is involved, to be quite strong. The 9 appearances she has made in the past year, have resulted in more than 40 print and broadcast placements about the Smoke-Free Class of 2000. On a national level, articles have appeared in such publications as Scholastic, Weekly Reader and NEA Today. Availability of the fifth grade materials and news of the bookcover contest are now being publicized.

7. Promotion of ALA, ACS, AHA School-based Materials

A key objective of the SFC2000 program is to encourage schools to obtain and use the more comprehensive tobacco use prevention programs available from the ALA, ACS and AHA. Each activity in the fifth grade materials incorporates a mention of each of these programs and the final page of the teacher's guide includes a full list of ALA, AHA, and ACS tobacco use prevention materials.
Many of the teachers that returned the evaluation forms either responded that they had requested more comprehensive materials or planned to do so.

8. Field Advisory Group

The Field Advisory Group, made up of volunteers and staff from ALA, ACS, and AHA, represents every region of the country. The purpose of this group is to provide guidance and recommendations to the project coordinator and the steering committee. This group was formed after the completion of the first SFC2000 evaluation and has met twice with a third meeting planned for April 1993. It continues to be a valuable resource. In 1991 the group developed the Strategic Plan for 1991-2000; in 1992 they organized and participated in a series of conference calls that involved the state coordinators for all 51 coalitions. These conference call focused on the national bookcover contest, the possibility of a mid-point activity such as a national conference in 1995, and other issues of mutual interest to the state coalitions. A great deal of problem solving and information exchange occurred during these conference calls.

Benefits

The SFC2000 program continues to be a winner because it collectively addresses the missions of ALA, ACS, AHA and positions each as a community agency that surmounts minor differences for the greater good, i.e., to help children learn to make healthy choices.

Volunteers involved in the program report a sense of pride when approaching local elementary schools with a program that represents the three agencies.

Public Policy initiatives such as the excise tax referendum in the state of Massachusetts are bolstered by the use of student representatives--youth ambassadors for a smoke free society in the year 2000.

The project open doors for ALA, ACS, AHA to promote more comprehensive school-based programs. A recent teacher survey found that 75% of the respondents said they would like to use educational materials offered by the three agencies.

Future Directions

It will be important to keep the momentum going over the next 7 years of the project's life. As the SFC2000 kids enter middle school, junior and senior high school and the pressure to smoke cigarettes, and to experiment with them will become greater and greater. In a like manner the program will be under increasing pressure to stay fresh and meaningful.

The Field Advisory Group, at its last meeting in April of 1992, recommended a mid-point conference in 1995 that not only brought together the SFC2000 volunteers and staff but also members of the SFC2000. This suggestion was supported 100% by the state coordinators during a series of conference calls held in May and June of 1992.

Other options for a mid-point activity include a collaboration between the local public policy staff and volunteers and the local SFC2000 staff and volunteers to focus on these youth ambassadors for a smoke free society as young advocates for a tobacco free 21st century. Finally, a media event to feature the midpoint accomplishments of the SFC2000 and the public policy efforts since 1988 has also been considered.

Nov. 18, 1992
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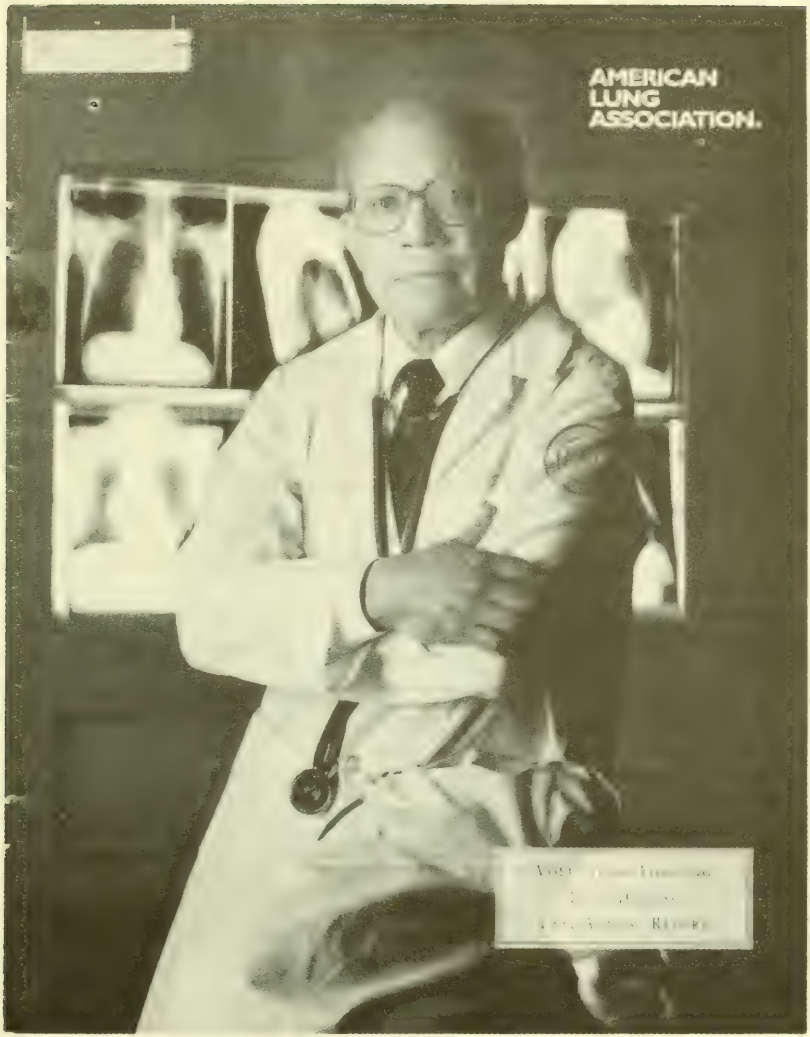
AMERICAN
LUNG
ASSOCIATION.

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Design and Graphics by John Kneapler Design NYC Writer: Susan Ince

The ALA thanks Mark Swisher, who for a second straight year has donated his skills as a photographer to this report. Mark's photos appear on the cover and pages 3 to 13. Thanks go as well to the many Lung Associations which nominated volunteers for the report's profile section. If cost weren't a factor, this book would be 100 pages long.

Having a child with asthma taught Jean Coffey what it's like to watch a loved one struggle for breath. But it also taught her that lung disease can be managed if you stay on top of it — a skill she passes on to parents who join the Family Asthma Program she started. Jean has also counseled parents whose babies survived respiratory distress syndrome (RDS). This lung disease once killed up to 25,000 infants a year, until Mary Ellen Avery came along. Dr. Avery, a pediatrician, found that babies she couldn't save lacked a substance she'd noticed in animal lungs. Her discovery of surfactant has helped cut RDS deaths by nearly 90 percent. Bob Neely isn't a doctor, but he's also done a lot for children's lungs. Bob lobbies at his own expense for laws that keep tobacco away from children. Such laws weren't on the books when John Prokopowicz took up smoking as a teen. John couldn't quit until emphysema made every breath he takes a conscious effort. That effort doesn't stop him from giving his time and telephone number to smokers who need support to quit. Nonsmokers can be stricken with lung disease too, as Gail Livingstone found when pulmonary fibrosis left her dependent on oxygen. Like the other Lung Association volunteers pictured here, Gail's experience only sharpened her commitment to helping others. She has organized support groups and writes about living with lung disease.

Good organizations give people the chance to achieve things they might never achieve on their own. The American Lung Association gives volunteers the means to fight lung disease. The resulting accomplishments are a source of tremendous pride to us, and a source of hope to the one in 10 Americans with chronic breathing problems.

On the following pages, we spotlight a small but representative group of our volunteers. We asked them to talk about their reasons for wanting to fight lung disease, and to say, in their own words, why they think this fight is important to all Americans.

The concerns they discuss may seem remarkably varied on the surface, but their underlying commitment springs from the same appreciation. As anyone who has ever experienced lung disease will tell you, *When you can't breathe, nothing else matters.*®

Growing numbers of Americans are experiencing this painful truth firsthand. In the 1980s, the prevalence of asthma rose by 53 percent, with the sharpest increases coming among children. Lung cancer and chronic bronchitis are up sharply, and tuberculosis is back with deadlier drug-resistant strains. Despite progress with conditions such as respiratory distress, breathing problems continue to be the No. 1 cause of death in American infants.

Joanna Fluros, Ph D

Homberto S Leal

Rebecca Bascom, M D

Charles P Felton, M D

Carolyn Martin

These alarming trends reinforce a conclusion researchers have been pointing to for years. The lung is a uniquely vulnerable organ. Our airways are directly exposed to pollutants ranging from ozone to secondhand smoke and infections ranging from TB to influenza.

The American Lung Association fights to reduce your risk of being exposed to these airborne hazards. Together with our 128 state and local Lung Associations, we raised $125 million last year for education, community service, research, and advocacy.

Donors today are increasingly eager to know what they really purchase through their donations. We urge you to read the following statements, which reflect the type of work being done by Lung Association volunteers across the country. Judge for yourself if their work is worth supporting. We're confident you'll be moved to agree that it is.

John R. Garrison
Managing Director

John D. White, Ph.D.
President, 1991-92

JOANNA FLOROS, PH.D.

When I was expecting my first child, I was naive as could be about the things that might go wrong. I trusted that everything would work out fine, and I was lucky. He was a healthy full-term baby.

It was only after I started doing research on lung development that I really came to understand how complicated birth is — especially the transition that babies make between floating in the liquid world of the womb and breathing air. Of all the breaths a person takes, a baby's very first is the most difficult.

"OF ALL THE BREATHS A PERSON TAKES, A BABY'S VERY FIRST IS THE MOST DIFFICULT."

Since a newborn starts with no air at all in the lungs, the effort needed to expand them is tremendous. You can get some sense of the challenge by seeing how much harder it is to blow up a brand-new balloon than one that has already been inflated once. Fortunately, most babies are able to make the transition smoothly. As pregnancy progresses, their lungs produce increasing amounts of surfactant, a detergent-like mixture of fats and proteins that helps air pockets stay open between each breath.

But when babies are born before enough surfactant has been produced, the result can be respiratory distress syndrome (RDS). Some babies can't even take their first breath, while others grunt or squeak with each inhalation. You can see how hard they're struggling to breathe by the way their nostrils flare and their rib muscles strain.

Babies with RDS are taken immediately from their mothers and brought to the neonatal intensive care unit. I got some idea of how painful this is for a mother when my second son was born. Like some other infants born by Caesarean section, a little fluid remained in his lungs after birth. He had signs of respiratory distress until it was finally cleared out after two days in the neonatal intensive care unit.

Today, most RDS babies are treated with a newly developed artificial surfactant, while others get surfactant derived from cow lungs. The majority need mechanical ventilation until they're ready to breathe on their own. Advances in neonatal medicine have pushed the survival rate up to 90 percent. For the tiniest babies — those weighing less than two pounds — nine in 10 used to die, but now half can be saved. Unfortunately, while mechanical breathing helps to keep babies alive, it can also scar tiny lungs and lead to lifelong breathing problems.

We still have a lot left to do, both in terms of saving the smallest preemies and in minimizing the time that RDS babies spend on mechanical ventilation. In my own research, I'm using the techniques of molecular genetics to learn more about the proteins in surfactant and about factors that interfere with surfactant production.

There's so much we still don't know. Why are boys more at risk for RDS? Why do some preemies do just fine, while others have severe problems? What genetic factors make RDS more frequent in certain families?

When we can better predict which babies are likely to develop RDS, and when we finally understand the essential steps that prepare the lungs for birth, I believe we can spare many infants and families from the trauma that RDS brings. It's very rewarding to think that my work may help to make that first breath as easy as possible.

More Babies Are Surviving RDS
Infant Deaths From RDS 1980-1990
5,000
4,500
4,000
3,500
3,000
2,500
80
82
84
86
88
90
Improvements in treating respiratory distress syndrome made possible in part by American Lung Association research, have greatly reduced infant deaths due to this condition.

HUMBERTO S. LEAL

Have you ever been in a hospital room where a patient was suffering respiratory failure? It's a scene I often confront in my work as a respiratory therapist — lung disease so out of control the patient needs mechanical help to breathe.

It's very satisfying to be the one who provides this help, and to watch normal coloring return to a person who was blue from lack of oxygen. But I've discovered an even greater source of fulfillment — teaching families how to provide care at home and avoid life-threatening crises.

An emphysema patient introduced me to the American Lung Association 10 years ago. He had been to an ALA Better Breathers club at another hospital, and he encouraged me to start a club here in the Rio Grande Valley. Now, we have support groups spreading out for a hundred miles.

Asthma groups are the most popular, especially groups for children. I really believe they save lives. Kids are far more likely to face severe consequences from asthma when families don't know the warning signs of a bad attack. I've seen cases where parents waited too long to rush to the hospital, and their beautiful child has died or suffered brain damage due to oxygen deprivation.

Asthma doesn't have to be life-threatening to ruin a kid's childhood. I know one boy, Mario, who was always going in and out of the hospital, missing weeks of school at a time. His mom was very devoted to him and his schooling, but no one had ever shown her how to respond to worsening asthma before a crisis. The kid was headed for failure at school and an isolated childhood.

> "ASTHMA DOESN'T HAVE TO BE LIFE-THREATENING TO RUIN A KID'S CHILDHOOD."

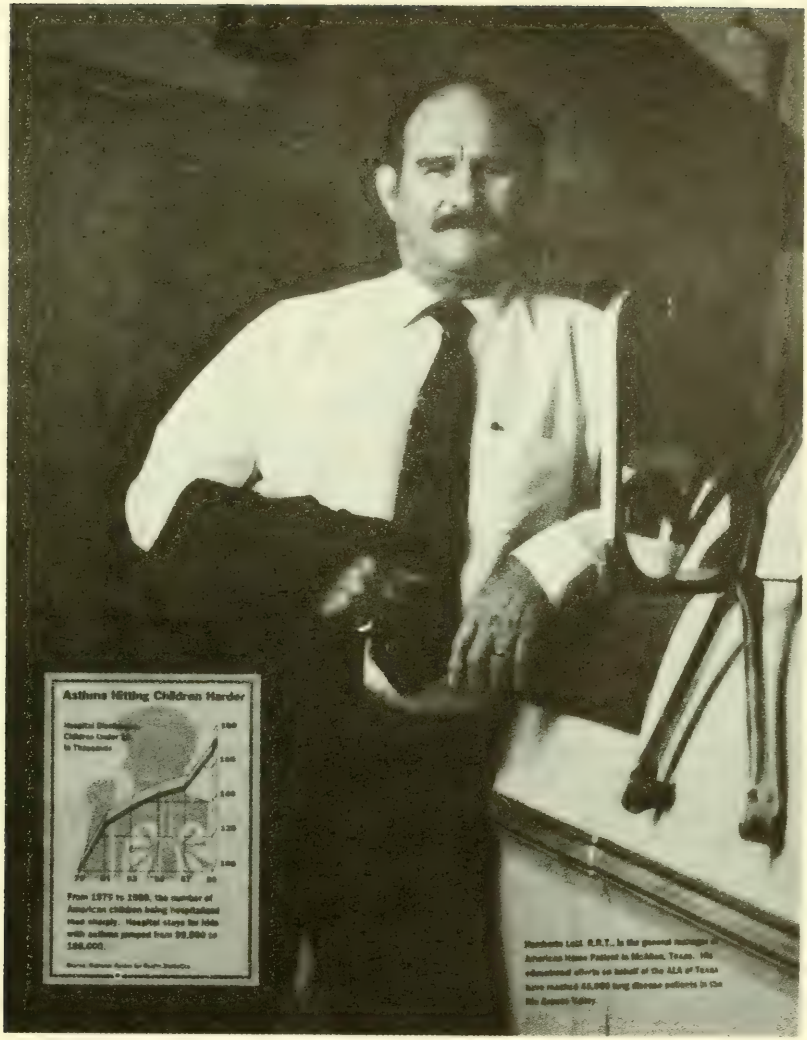
Then we started an asthma support group in his area, and Mario and his mother signed up. His teachers can finally see how bright he is, and he's well enough to play football with his friends. He even volunteers at our support group, telling other kids how controlling his asthma helped turn his whole life around.

Not all the kids I see have asthma as bad as Mario's, but even mild asthma can get kids in trouble. When the disease isn't recognized, children get blamed for their symptoms. Parents call them cranky or lazy. Teachers label them moody or disruptive.

Then you have the opposite problem of parents smothering their children because they have asthma. The family treats the kid like he's disabled, which is the worst thing you can do. Besides spoiling all the fun, not exercising can actually make a child feel much worse.

I'm a strong believer in children with asthma knowing all they can about their condition. I know kids who are real pros at juggling their medications and using peak flow meters to spot attacks before they even start wheezing. Some will never outgrow the disease, but by learning to make asthma a routine part of life, they can accomplish pretty much the same thing.

When the Olympics were on last year, it was so exciting to see it dawn on these kids that their goals could go much higher than staying well and out of the hospital. On TV, they saw champions using inhalers before swimming or running. And for the first time, some of them realized, "I can do that, too!"


Asthma Hitting Children Harder
From 1979 to 1988, the number of American children being hospitalized rose sharply. Hospital stays for kids with asthma jumped from 99,000 to 169,000.

Rebecca Bascom, M.D.

"Show me a dead body." That's the kind of proof some people want before they can be persuaded that air pollution is really a threat.

But research shows air pollution can be lethal. When pollution levels are high, more patients are hospitalized, and more patients die.

As a lung doctor, I don't need a radio to hear air quality reports; I can get them from my waiting room. Take Gail, for example. She's a well-conditioned tennis player who's normally very good at controlling her asthma — but not in the summer when air pollution soars. Then there's John, a veteran with lung disease, who won't even consider exercise in the summer, because it hurts to breathe when he goes outdoors.

> "You can buy bottled water, but you can't buy bottled air."

The problems I see aren't limited to people with lung disease. It isn't hard to find what I call pollution's "walking wounded" among the healthy. A woman thinks she has a summer cold, but it's really a reaction to pollution. An athlete can't finish his workout because he's short of breath and feeling tight in the chest. He thinks he's getting out of shape; what he's really getting is a lungful of ozone.

I try to explain to my patients that their lungs have a challenging job. Every day, they have to process 10,000 liters of air, absorbing the oxygen the body needs while keeping out all the bad things mixed in with it.

In the lab, we're able to document pollution's assault on our lungs. Sulfur dioxide sends airways into spasm. Ozone triggers a chemical cascade inside the airways, causing a reaction that one researcher compared to a sunburn. White blood cells accumulate and proteins leak from the damaged tissues. It's the same reaction you see when you scrape a finger, and the skin becomes swollen and weepy.

In my state, nine in 10 people are exposed to ozone levels that exceed federal safety standards. The media calls it a city problem, but the highest ozone levels are actually in the state's rural northeast corner. I know health-conscious people who buy only bottled water and move their families 80 miles from the city. They think they're escaping pollution, but they're not. You can buy bottled water, but you can't buy bottled air.

We're unlikely to get cleaner air without taking a serious look at our cars and suburban sprawl. Nationally, 58 percent of carbon monoxide pollution and 23 percent of ozone can be blamed on motor vehicles.

I always encourage people to live near the place they work, to reduce the miles they drive, and to minimize the escape of gasoline vapors by not topping off their gas tanks. Every little bit helps, but real progress depends on ensuring all cars run as cleanly as technology can make them. The Lung Association is urging states to adopt low-emission vehicle programs. California, New York, and Massachusetts have taken that step, and a dozen states are following their lead. We also need to plan our growth to make transit less of a problem.

Experience has shown me that health isn't only an individual concern. The environment you live in makes a difference; it can either preserve your health or damage it. That's why I decided to volunteer for the Lung Association, talking to legislators and the public about clean air issues. To me, fighting air pollution is simply another way to practice good medicine. It's also a legacy for my children.

Can Air Pollution Kill?
% Increase in Death Rates Caused by Increased Airborne Particles*
19%
14%
All Deaths
Cardiovascular Disease
Pneumonia
Chronic Lung Disease

CHARLES P. FELTON, M.D.

Thirty-six years ago, I walked into an old-fashioned hospital ward — 60 beds in one room and each bed filled with a seriously ill patient. Many of these patients didn't survive, and those who did needed months of care before being discharged or transferred to sanitariums.

It was a stark introduction to tuberculosis. Still, I was optimistic. New medicines had just become available, and the conquest of the disease seemed to be within sight.

Now, Americans have awakened to a resurgence of TB. The patients are harder to treat than those we saw in the 1950s, while our public health system has become so financially crippled, the resources we enjoyed back then seem bountiful by comparison.

What happened? I believe too many people claimed victory too soon and forgot how resilient tuberculosis can be.

"WITHOUT SWIFT ACTION, I PERSONALLY BELIEVE NEW TB CASES WILL DOUBLE IN FIVE YEARS."

When you first become infected with TB, you can't actually feel the germ attacking your lungs. If you're well-nourished and have a healthy immune system, your body can wall off damaged tissue to keep the infection from spreading. If you're not so well-fortified, the germ breaks through every barrier you create, leaving you with less and less healthy lung. We hardly ever use the old term "consumption," but it's accurate. With TB, your lungs are literally consumed.

Fortunately, if you're diagnosed and conscientiously take your medicine, you can almost always prevent tuberculosis from progressing. The problem is, many patients today aren't good at taking pills for months at a time. Most are facing crises that make TB seem less urgent by comparison. Where you have drug abuse, you have TB. Where you have homelessness, you have TB. Where you have AIDS, you have TB.

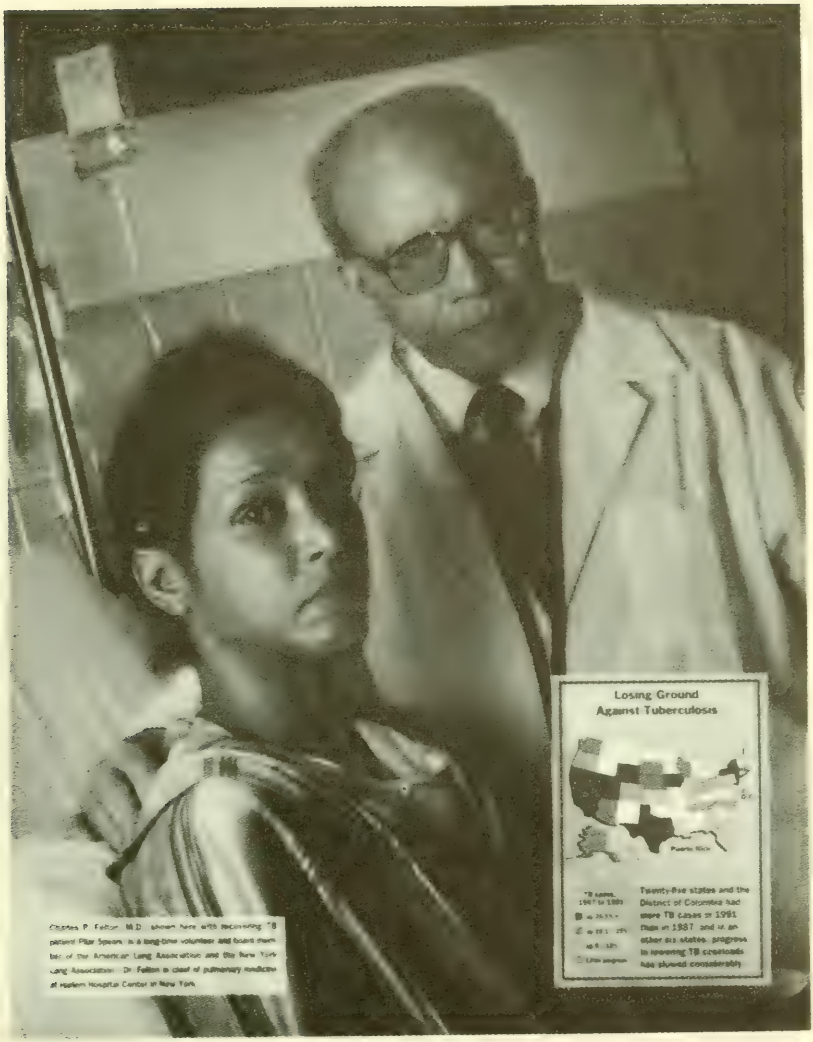
Unless you provide supervised therapy for these patients, many stop taking their pills as soon as they feel better. Since their tuberculosis isn't really cured, it relapses, and the germs that are responsible for the relapse may be resistant to standard antibiotics. The most hair-raising thing we're seeing now is TB that's resistant to four or more different drugs. These infections are often deadly and can be passed along to others.

Tuberculosis has become a major problem in the nation's largest cities, but it's also on the rise in places like Wisconsin, San Diego, and my home town of New Orleans. And it's not just homeless people or people with AIDS who are getting infected. Nationwide, there has been a dramatic rise in TB among children. More people in the health care and social service industries are testing positive. In Maine, one factory worker infected hundreds of co-workers before being diagnosed.

The threat is truly ominous. Without swift action, I personally believe new cases will double in the next five years.

We need resources to monitor patients' therapy and to trace their contacts so they can be tested and treated. In the '50s, we had the National Tuberculosis Association — now the American Lung Association — watching health departments to make certain they fulfilled their responsibility. TB is once again among the ALA's top priorities. Last year, our expert testimony secured a $58 million increase in federal TB funds.

We tell Congress — and anyone who will listen — it's not too late to stop the growing TB epidemic. If we have the will to fight it, TB can still be conquered.


Losing Ground
Against Tuberculosis
Puerto Rico
Twenty-five states and the District of Columbia had more TB cases in 1991 than in 1987, and in another six states, progress in lowering TB caseloads has slowed considerably.

Charles P. Felton, M.D., shown here with recovering TB patient Pilar Spears, is a long-time volunteer and board member of the American Lung Association and the New York Lung Association. Dr. Felton is chief of pulmonary medicine at Harlem Hospital Center in New York.

CAROLYN MARTIN

If you've been in California lately, you may have heard this joke on the radio: Why sell cigarettes? Why not just sell phlegm and cut out the middle man? The teens I know think that's hilarious. They also like the TV ads featuring a rap star singing, "They brought us to pick it, now they want us to smoke it."

Surveys show that two-thirds of kids in California can remember these messages, which were written with a youth audience in mind. More importantly, they're taking the message to heart by quitting or refusing to ever start smoking. In just two years, the number of California smokers in the 4th to 12th grades dropped by 37,000.

All this is making tobacco companies nervous. They need to recruit 4,750 new smokers each day to replace current smokers who die or quit. It's very difficult to get adults to start — they already know too much — so cigarette makers are targeting teens and even kids in grade school. They claim they're just trying to get people to switch brands, but that's nonsense. They know perfectly well that half of all smokers start by age 14, and nine in 10 begin by the time they're 20.

How do they target kids? The Virginia Slims campaign is a good example. They didn't pull that name out of thin air; they deliberately set out to snag young women who were concerned about their weight. Other cigarette ads convey that smoking will make you sexy and help you relax. Joe Camel's cool party-loving image appeals especially to 12- to 13-year-olds.

We used to beg local community groups for enough money to reach even one in 10 children with classroom education about tobacco. Now, there's money to reach every kid in the state, thanks to an initiative, Prop 99, which voters approved in 1988. It increased the tax on cigarettes by 25 cents a pack and earmarked 20 percent of the revenue for anti-smoking education. In addition to school programs, Prop 99 pays for a media campaign and all kinds of creative activities that counter tobacco industry propaganda.

> "I DIDN'T THINK IT WAS WEIRD THAT MY FATHER NEEDED A CIGARETTE BEFORE HE COULD GET OUT OF BED. KIDS TODAY KNOW BETTER."

Passing Prop 99 wasn't easy. When we tried the state legislature, we couldn't get a single vote in the health committee. During the debate, one committee member paced the aisles smoking!

That's why we decided to take the initiative directly to the people. The Lung Association gathered two-thirds of the volunteer-generated signatures needed to put Prop 99 on the ballot. Working with other groups, we raised $1.6 million to support the measure — against $20 million spent by the tobacco lobby to fight us. Yet we still won.

Since the education program started in 1991, half a million Californians have stopped smoking. The cigarette companies are getting desperate; they're trying to buy off state and local legislators with campaign contributions larger than the gifts they usually make to members of Congress. The struggle isn't over, but I don't think the industry can stop us now.

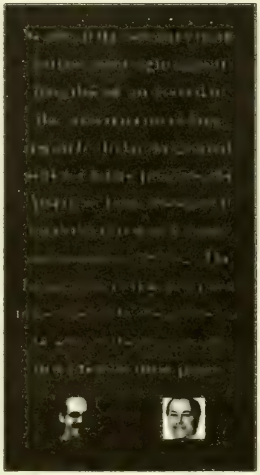
We've made truly historic progress in changing the way people think about smoking. I remember when I was little, I didn't think it was weird that my father needed to smoke a cigarette before he got out of bed in the morning. Kids today know better.

Tobacco and Substance
Abuse Among Teens
Smokers
Non-Smokers
Drug
Drinking
Marijuana
Teens who smoke are more likely to
engage in other unhealthy behaviors
than teens who don't smoke. Smoking
prevention keeps the risk that teens tell
them it's OK to use drugs and alcohol.

Research

While nearly all Americans know about AIDS, few have heard of an even larger killer known as ARDS. This lung disease, adult respiratory distress syndrome, kills half or more of the 150,000 Americans it strikes annually.

ARDS usually arises as an aftereffect of another major trauma, such as drug overdose or injuries from a car crash. In patients stricken with the syndrome, the air sacs collapse and fill with fluids. This severely restricts the flow of life-giving oxygen into the body, and ARDS patients must receive additional oxygen to survive. The irony is that high doses of oxygen also injure the lung by producing substances known as free radicals.



"It's a terrible bind," says Sadis Matalon, Ph.D., a professor at the University of Alabama at Birmingham School of Medicine. "Free radicals are very toxic with an effect that is similar to radiation. Normally, the lung has enzymes that detoxify free radicals, but if you get too much oxygen, these defenses are overwhelmed."

Dr. Matalon is pursuing a new and promising approach to ARDS: surfactant therapy. A lack of surfactant causes respiratory distress in premature newborns, and loss of this soap-like substance in the lung may also be critical in adult respiratory distress.

"We've shown that if you give surfactant prophylactically to animals, you limit the lung injury caused by excess oxygen," says Dr. Matalon. "In humans, the main hurdle is finding ways to get the surfactant where it's needed. It's a tough challenge, but we think it's the only way to make a major impact on the syndrome."

Lung doctors are playing a leading role in the battle to lengthen the lives of people with AIDS. Their involvement stems from the need to treat the numerous lung infections common in AIDS.

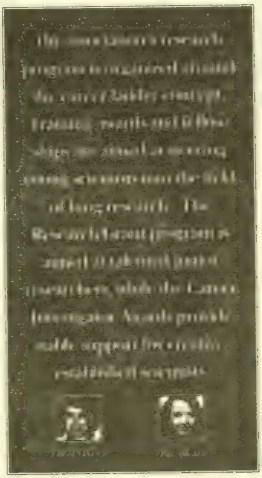
Chief among these is *Pneumocystis carinii* (PC), a normally harmless organism that can kill individuals with damaged immune systems. "Even with preventive treatment, 20 to 40 percent of AIDS patients get pneumocystis pneumonia," says Dr. Jay Fishman, an assistant professor of Medicine at Harvard Medical School. "Half these patients can't tolerate our current antibiotics due to side effects, which is why PC is still a significant cause of pneumonia and death in AIDS."

To give AIDS patients more options, Dr. Fishman is searching for new drugs to treat PC. His search is focused on a group of antibiotics, including a class known as quinolones, which can kill the *Pneumocystis* germ by blocking the action of a key enzyme called topoisomerase II.

"There are many quinolones," says Dr. Fishman, "and we can't tell just by looking at them which ones are most effective against PC. Since all drugs have some toxicity, you want to find the best ones possible to limit side effects and maximize efficacy."

Dr. Fishman's present goal is to clone the gene for topoisomerase. This in turn would enable researchers to make large quantities of the enzyme, which would greatly speed the search for drugs to block it. The best drug, when found, would benefit not only AIDS patients but those with weakened immune systems due to transplant surgery and cancer chemotherapy.

Not everyone with emphysema gets the disease from smoking. Some 50,000 to 100,000 Americans are genetically predisposed to develop this lethal lung disease because they can't produce enough of a protein known as alpha 1-antitrypsin (AAT). Depending on the severity of the defect, people with AAT deficiency can start to develop emphysema in their 30s.



Dr. Katherine Ponder's goal is to cure this disease through gene therapy (the replacement of faulty or missing genes with healthy genetic material). "We've had some exciting results," says Dr. Ponder, an assistant professor at Washington University School of Medicine in St. Louis. "We've been able to put the gene for making AAT into rats, and these transplanted genes have expressed themselves consistently for months. That's a very important step."

The challenge, says Dr. Ponder, is getting the modified cells to produce enough AAT. "Right now, we can get the gene into just one percent of the cells we're targeting. One obvious way of overcoming this problem is to get these modified cells to make AAT at 100 times the normal level."

Toward this end, Dr. Ponder is studying "promoters" — genes that signal other genes to express themselves at high levels. By packaging the gene for AAT with the right promotor, it may be possible to achieve therapeutic levels of AAT production.

"We're not there yet," says Dr. Ponder, "but we think we're on the right track. We may be ready for human trials of this therapy in as little as two years."

"The sharp rise in asthma deaths and illness among black children in the inner city is a serious public health problem," says Shari Wade, Ph.D. Dr. Wade, who has asthma herself, is studying factors that might explain the increase in this common lung disease.

As a pediatric pathologist at Rainbow Babies and Children's Hospital in Cleveland, Dr Wade is following a group of 72 African-American children with moderate to severe asthma. She says, "We're finding that kids with fewer social resources — supportive friends and family — are more likely to have been hospitalized for asthma. This shows there are social aspects to the disease we need to address."

One clear reflection of family problems is conflicts about medication. Hospital stays are more common, says Dr. Wade, in cases where children resist taking medicine and parents aren't able to overcome that resistance. "Some kids throw tantrums," she says, "but the problem is mostly passive resistance. They make excuses or wander away when it's time to take their medicine."

To give parents the skills to cope with childhood asthma, Dr. Wade is using her findings to help design more effective prevention programs. "As part of the study, we're recruiting people with social service backgrounds to do home visits. These are community residents with good parenting skills who know how to handle difficult children and access community resources. We give them training in asthma, and they go out and help mothers try to solve the problems they're having."

Fund Raising

Traditionally, fund raising for the American Lung Association could be summarized in two words: Christmas Seals.® The Christmas Seal Campaign® is still a vital source of funds for fighting lung disease, accounting for nearly $35 million of the $60 million raised last year through direct mail appeals. However, as the Lung Association's mission has broadened from tuberculosis to all lung disease, its fund-raising focus has also broadened to embrace a range of activities.

- In 1991-92, nationwide revenue from special events increased by 11 percent to $14 million. Bike events, including the Clean Air Challenge,™ raised $4 million, and proceeds from the Golf Privilege Card® also approached $4 million.
- Bequests to Lung Associations totaled $8.7 million, a 10 percent increase over the previous year. Over 60 Lung Associations now have Heritage Clubs to recognize donors who have pledged to fight lung disease through a bequest or other planned gift.
- The volunteer corporate advisors who serve on the ALA's President's Council (listed below) were responsible for program grants and sponsorships totalling more than $1 million last year.
- In the largest corporate partnership in its history, the Lung Association joined with CIBA-GEIGY Corporation to distribute over $7 million worth of educational materials aimed at helping smokers to quit.
- The Ninth Annual Research Fund Dinner was the most successful to date, raising $233,000. At the VIP reception before the dinner, the Basel Pharmaceuticals Division of CIBA-GEIGY Corporation and Searle received the Founder's Award, which is given to recognize cumulative gifts of $500,000 or more.
- Many ALA programs are made possible through generous corporate and foundation underwriting. Supporters in 1991-92 included CIBA-GEIGY Corporation (smoking cessation and African-American lung health); Searle (smoking cessation); Fisons Pharmaceuticals and the National Rural Letter Carriers (asthma education); Pfizer Laboratories (adult lung disease); the Robert Wood Johnson Foundation (occupational lung disease); Boehringer Ingelheim Pharmaceuticals, Inc. (adult lung disease, smoking cessation, and research); Connaught Laboratories, Inc., Miles, Inc., and Parke-Davis, Division of Warner-Lambert (flu and pneumonia prevention); Genentech, Inc., and Schering Corporation (lung research); and the Nathan Cummings Foundation (African-American lung health).
- ALA's partnership with SmithKline Beecham Consumer Brands provided major support through a "Season For Giving" cause-related marketing promotion.
- Employee giving at the workplace increased by 20 percent to $3.7 million, primarily on the strength of partnerships with local United Ways and Combined Health Appeals.

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Help Us Fight Lung Disease

There are many ways you can support the work of the American Lung Association through gifts of cash, securities, real estate, and personal property. In addition to benefiting the fight against lung disease, there are certain forms of "planned giving" which offer benefits to the giver. A planned gift is one which you arrange during your lifetime whose principal value does not accrue to the American Lung Association until some future time. In many instances, these gifts provide the donor with significant tax advantages.

WILLS AND BEQUESTS. Bequests may be made to any Lung Association. The following form may be used for a bequest: "I give and bequeath to the American Lung Association, a nonprofit corporation,

A) all the rest and residue of my estate, (or)
B) ____ percent of my estate, (or)
C) the sum of ____ dollars, (or)
D) the following assets or property [described here] for its general purposes."

An unlimited estate tax deduction is allowed for gifts of cash or other property bequeathed to the Lung Association in your will.

"Planned giving helps donors to help themselves while advancing the fight against lung disease."

GIFT ANNUITY. In exchange for your irrevocable gift, the American Lung Association agrees to pay you a fixed dollar amount during your life and/or the life of a designated loved one. The amount and tax benefits you receive are determined by the size and type of your gift, your age, and the age of any other beneficiary. Your income is guaranteed by the American Lung Association and is not subject to market fluctuation.

ANNUITY TRUST. Through an annuity trust, you transfer cash or other assets into an irrevocable trust and direct that a fixed dollar amount be paid to you as long as you live. At your death, the same fixed income can be paid to any surviving loved ones, as long as they live. The principal cannot be used by any Lung Association until these conditions have been met.

UNITRUST. This gift works in much the same way as the annuity trust except for a flexible income feature that sometimes can be a cushion against inflation. Instead of a fixed dollar amount to the beneficiaries, the trust agreement specifies a percentage of the trust's fair market value as determined annually.

POOLED INCOME FUND. Your assets are "pooled" into a large combined investment fund similar to a mutual fund. All the net income earned by the fund is distributed to the beneficiaries based on their number of "units" in the fund. At the death of the last income beneficiary, the remainder is used to fight lung disease by the Lung Association of your choice.

REVOCABLE TRUST. Not everyone is financially or emotionally ready to irrevocably assign assets to a trust, but with a revocable trust, you can get your assets back. This agreement can provide income to you and your spouse for life with the remainder going to the Lung Association at your death. It is the most flexible form of trust and can be funded with a variety of assets.

LIFE INSURANCE. The American Lung Association is named owner and/or beneficiary of an existing policy that is no longer needed. It is an opportunity to make a more substantial contribution than might otherwise be possible.

REAL ESTATE. A gift of real estate may be made outright to the American Lung Association; it may also be made to fund a charitable remainder unitrust, or made under a life estate agreement, where the donor retains the right to live in the property for life and gets current tax benefits.

For more information on these planned giving opportunities, contact your local American Lung Association listed in the white pages of your telephone book, or write to Manager, Gift Planning, American Lung Association, 1740 Broadway, New York, NY 10019-4374.

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INDEPENDENT AUDITOR'S REPORT

LOEB & TROPER
Certified Public Accountants
270 Madison Avenue, New York, NY 10016

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AMERICAN LUNG ASSOCIATION

We have audited the accompanying consolidated balance sheet of American Lung Association and its constituent and affiliate Lung Associations as of June 30, 1992, and the related consolidated statements of support, revenue, expenses and changes in fund balances, and functional expenses for the year then ended. These financial statements are the responsibility of the Association's management. Our responsibility is to express an opinion on these financial statements based on our audit. We did not audit the financial statements of the constituent and affiliate Lung Associations, which statements reflect total assets as of June 30, 1992, of $134,263,565 and total revenue of $109,703,021 of the related consolidated totals. These statements were audited by other auditors, whose reports thereon have been furnished to us, and our opinion expressed herein, insofar as it relates to the amounts included for the constituent and affiliate Lung Associations, is based solely upon the reports of the other auditors.

We conducted our audit in accordance with generally accepted auditing standards. Those standards require that we plan and perform the audit to obtain reasonable assurance about whether the financial statements are free of material misstatement. An audit includes examining, on a test basis, evidence supporting the amounts and disclosures in the financial statements. An audit also includes assessing the accounting principles used and significant estimates made by management, as well as evaluating the overall financial statement presentation. We believe that our audit provides a reasonable basis for our opinion.

American Lung Association and Constituent and Affiliate Associations
CONSOLIDATED BALANCE SHEET JUNE 30, 1992 (With comparative totals for 1991)

	CURRENT FUNDS					TOTAL	
ASSETS	UNRESTRICTED	DONOR RESTRICTED	LAND, BUILDING, AND EQUIPMENT FUND	ENDOWMENT FUND	ANNUITY GIFT FUND	1992	1991 (NOTE 9)
Current assets							
Cash (principally interest-bearing accounts)	$ 23,046,623	$1,599,063	$ 91,437	$ 181,558	$ 9,718	$ 24,928,399	$ 20,488,956
Accounts receivable	3,754,492	1,331,841	5,480			5,091,813	3,350,280
Short-term investments – at cost (approximates market)	20,213,121	716,356	367,318	863,633	538,528	22,698,956	24,517,618
Inventory of educational and fund-raising materials	3,831,147					3,831,147	3,512,387
Prepaid expenses and other current assets	5,050,873	243,150	953	20,000		5,314,976	5,760,966
Notes and loans receivable – current portion	669,594	33,361				702,955	1,194,688
Interfund receivables (payables)	(3,085)	(166,288)	2,072	167,301			
TOTAL CURRENT ASSETS	56,562,765	3,757,483	467,260	1,232,492	548,246	62,568,246	58,824,895
Noncurrent assets							
Investments – at cost (market value of $74,664,984 in 1992 and $77,974,429 in 1991) (Note 4)	53,667,681	570,830		11,825,605		66,064,116	71,359,944
Fixed assets (net of accumulated depreciation and amortization of $19,145,126 in 1992 and $17,407,695 in 1991) (Note 3)			26,996,131			26,996,131	29,038,797
Notes and loans receivable – long-term portion	638,442		23,273			661,715	376,857
Other noncurrent assets	613,332	75,729				689,061	569,398
TOTAL NONCURRENT ASSETS	54,919,455	646,559	27,019,404	11,825,605		94,411,023	101,344,996
TOTAL ASSETS	$111,482,220	$4,404,042	$27,486,664	$13,058,097	$548,246	$156,979,269	$160,169,891
LIABILITIES AND FUND BALANCES							
Current liabilities							
Accounts payable and other current liabilities	$ 9,198,556	$ 317,444	$ 11,581	$ 1,702		$ 9,529,283	$ 11,862,318
Current portion of long-term debt (Note 5)	126,085		1,095,306			1,221,391	1,117,121
Current portion of deferred revenue	2,966,506	1,341,530	2,563			4,310,599	3,041,727
TOTAL CURRENT LIABILITIES	12,291,147	1,658,974	1,109,450	1,702		15,061,273	16,021,166
Noncurrent liabilities							
Long-term debt (Note 5)			3,081,781			3,081,781	4,394,784
Deferred revenue – long-term portion	1,248,950	172,026			$548,246	1,969,222	1,799,155
Other noncurrent liabilities	1,851,345					1,851,345	1,218,844
TOTAL NONCURRENT LIABILITIES	3,100,295	172,026	3,081,781		548,246	6,902,348	7,412,783
TOTAL LIABILITIES	15,391,442	1,831,000	4,191,231	1,702	548,246	21,963,621	23,433,949
FUND BALANCES	96,090,778	2,573,042	23,295,433	13,056,395		135,015,648	136,735,942
TOTAL LIABILITIES AND FUND BALANCES	$111,482,220	$4,404,042	$27,486,664	$13,058,097	$548,246	$156,979,269	$160,169,891

See independent auditor's report.
The accompanying notes are an integral part of these statements.

In our opinion, based on our audit and the reports of the other auditors, the aforementioned financial statements present fairly, in all material respects, the financial position of American Lung Association and its constituent and affiliate Lung Associations as of June 30, 1992, and the results of its operations for the year then ended in conformity with generally accepted accounting principles.

As discussed in note 10 to the financial statements, one of the affiliates acts as trustee, or has been named as successor trustee, for various trusts. These trusts have not been audited. An audit of these trusts by an independent certified public accountant has commenced. The ultimate findings of the audit cannot presently be determined. Accordingly, no provision for any liability that may result from the findings of the audit has been made in the accompanying financial statements.

Laab & Tryon

CERTIFIED PUBLIC ACCOUNTANTS
September 30, 1992, except for Note 9, as to which the date is November 23, 1992, and Note 10, as to which the date is March 1, 1993.

American Lung Association and Constituent and Affiliate Associations

CONSOLIDATED STATEMENT OF SUPPORT, REVENUE, EXPENSES AND CHANGES IN FUND BALANCES YEAR ENDED JUNE 30, 1992 (With comparative totals for 1991)

	CURRENT FUNDS				TOTAL	
PUBLIC SUPPORT AND REVENUE	UNRESTRICTED	DONOR RESTRICTED	LAND, BUILDING, AND EQUIPMENT FUND	ENDOWMENT FUND	1992	1991 (NOTE 9)
Public support						
Contributions	$ 69,634,338	$1,548,930	$ 25,358	$ (97,974)	$ 71,110,652	$ 74,869,974
Bequests	8,053,497	721,969	21,375	115,729	8,912,570	8,310,737
Special events (net of direct expenses of $5,179,288 in 1992 and $4,730,739 in 1991)	13,169,622	385,580	371,674	1,177	13,928,053	12,560,557
TOTAL PUBLIC SUPPORT	90,857,457	2,656,479	418,407	18,932	93,951,275	95,741,268
GRANTS FROM GOVERNMENTAL AGENCIES		3,770,567			3,770,567	4,702,637
Revenue						
Investment income	5,821,700	136,033	7,774	620,148	6,585,655	8,067,832
Gain (loss) on sale of investments	1,071,839		(1,585)	597,767	1,668,021	462,303
Service fees	6,222,111				6,222,111	5,493,100
Membership dues	1,315,829				1,315,829	1,023,487
Sale of materials	7,432,834				7,432,834	3,290,464
Other revenue	4,117,453	139,953	(231,797)		4,025,609	4,197,874
TOTAL REVENUE	25,981,766	275,986	(225,608)	1,217,915	27,250,059	22,535,060
TOTAL PUBLIC SUPPORT AND REVENUE	116,839,223	6,703,032	192,799	1,236,847	124,971,901	122,978,965
EXPENSES						
Program services						
Environmental health	12,328,129	1,491,503	324,205	231	14,144,068	12,051,722
Smoking education	21,856,504	2,212,616	427,837	231	24,497,188	23,971,167
Lung disease	34,938,307	2,325,292	678,578	4,546	37,946,723	35,227,828
Community health services	20,762,218	256,319	491,554	392	21,510,483	20,884,626
TOTAL PROGRAM SERVICES	89,885,158	6,285,730	1,922,174	5,400	98,098,462	92,135,343
Supporting services						
Management and general	7,344,277	20,348	291,603	198	7,656,426	8,105,502
Fund raising	20,402,664	34,213	282,690		20,719,567	21,441,649
TOTAL SUPPORTING SERVICES	27,746,941	54,561	574,293	198	28,375,993	29,547,151
TOTAL EXPENSES	117,632,099	6,340,291	2,496,467	5,598	126,474,455	121,682,494
EXCESS (DEFICIENCY) OF REVENUE OVER EXPENSES	(792,876)	362,741	(2,303,668)	1,231,249	(1,502,554)	1,296,471
Other changes in fund balances						
Equipment acquisitions financed by other funds	(1,564,542)	(43,740)	1,608,282			
Debt repayment financed by other funds	(325,955)		325,955			
Other transfers and adjustments	963,841	(47,590)	(688,856)	(445,135)	(217,740)	(411,497)
FUND BALANCES – BEGINNING OF YEAR – AS ORIGINALLY STATED	97,696,873	2,714,090	24,452,434	12,270,281	137,333,678	136,184,881
PRIOR PERIOD ADJUSTMENTS (NOTE 9)	113,437	(412,459)	(298,714)		(597,736)	(333,833)
FUND BALANCES – BEGINNING OF YEAR – RESTATED	97,810,310	2,301,631	24,353,720	12,270,281	136,735,942	135,850,968
FUND BALANCES – END OF YEAR	$ 96,090,778	$2,573,042	$23,295,433	$13,056,395	$135,015,648	$136,735,942

See independent auditor's report.

The accompanying notes are an integral part of these statements.

American Lung Association and Constituent and Affiliate Associations
CONSOLIDATED STATEMENT OF FUNCTIONAL EXPENSES YEAR ENDED JUNE 30, 1992 (With comparative totals for 1991)

	PROGRAM SERVICES					SUPPORTING SERVICES			TOTAL	
	ENVIRONMENTAL HEALTH	SMOKING EDUCATION	LUNG DISEASE	COMMUNITY HEALTH SERVICES	TOTAL	MANAGEMENT AND GENERAL	FUND RAISING	TOTAL	1992	1991 (NOTE 9)
Salaries	$ 5,687,389	$ 9,291,460	$12,502,899	$ 9,186,063	$36,667,811	$3,644,798	$ 4,517,659	$ 8,162,457	$ 44,830,268	$ 42,794,490
Employee health and retirement benefits	728,286	1,183,876	1,579,165	1,129,312	4,620,639	448,184	526,727	994,931	5,615,570	5,389,745
Payroll taxes	481,224	774,538	1,061,483	781,560	3,098,805	307,421	364,447	671,868	3,770,673	3,611,431
TOTAL SALARIES AND RELATED EXPENSES	6,896,899	11,249,874	15,143,547	11,096,935	44,387,255	4,420,423	5,408,833	9,829,256	54,216,511	51,795,666
Professional fees and contract payments	1,454,059	1,943,093	2,696,566	1,396,267	7,489,985	1,020,397	3,525,252	4,545,649	12,035,634	12,486,546
Insurance	117,415	207,419	299,047	201,284	825,165	96,670	91,725	188,395	1,013,560	893,886
Building occupancy	805,412	1,125,334	1,661,541	1,223,103	4,815,390	472,595	532,880	1,005,475	5,820,865	5,538,569
Telephone	223,596	376,131	511,510	379,476	1,490,713	119,980	152,908	272,888	1,763,601	1,734,377
Travel and local transportation	235,634	333,401	488,044	502,406	1,559,485	80,498	160,146	240,644	1,800,129	1,714,468
Meetings and conferences	552,625	640,566	1,838,562	997,732	3,829,485	253,265	227,177	480,442	4,309,927	4,064,638
Supplies	165,083	236,329	397,929	236,824	1,036,165	81,788	121,978	203,766	1,239,931	1,191,462
Printing, publishing, and audiovisual materials	1,841,141	5,526,908	4,345,517	2,107,504	13,821,070	244,674	5,835,541	6,080,215	19,901,285	18,089,831
Postage and shipping	878,285	1,536,790	2,093,449	1,387,967	5,896,491	221,219	3,942,109	4,163,328	10,059,819	9,061,930
Media expenses	61,709	69,620	99,390	113,394	344,113	13,338	106,227	119,565	463,678	711,940
Office equipment	37,049	80,595	105,645	76,496	299,785	17,620	34,296	51,916	351,701	471,367
Repairs, rentals, and equipment maintenance	148,599	160,400	306,662	164,128	779,789	77,664	80,052	157,716	937,505	899,839
Dues and subscriptions	37,486	43,416	66,226	77,888	225,016	18,496	24,771	43,267	268,283	257,354
Awards and grants	73,524	237,990	5,726,678	653,458	6,691,650				6,691,650	6,820,857
Support of other organizations	79,958	246,787	421,026	86,291	834,062	32,514	40,472	72,986	907,048	824,167
Assistance to individuals	40,284	18,699	598,995	39,582	697,560				697,560	542,799
Miscellaneous	171,105	235,999	467,811	278,194	1,153,109	193,682	152,510	346,192	1,499,301	2,052,785
TOTAL EXPENSES BEFORE DEPRECIATION AND AMORTIZATION	13,819,863	24,069,351	37,268,145	21,018,929	96,176,288	7,364,823	20,436,877	27,801,700	123,977,988	119,152,681
Depreciation and amortization	324,205	427,837	678,578	491,554	1,922,174	291,603	282,690	574,293	2,496,467	2,529,813
TOTAL EXPENSES	$14,144,068	$24,497,188	$37,946,723	$21,510,483	$98,098,462	$7,656,426	$20,719,567	$28,375,993	$126,474,455	$121,682,494

See independent auditor's report. The accompanying notes are an integral part of these statements.

American Lung Association and Constituent and Affiliate Associations
NOTES TO FINANCIAL STATEMENTS JUNE 30, 1991

NOTE 1 – DESCRIPTION OF ORGANIZATION

American Lung Association National Office (ALA) together with its constituent and affiliate Lung Associations (hereafter referred to as the Association) are not-for-profit voluntary health agencies dedicated to the fight against lung disease through programs of public education on environmental health, smoking, lung disease, and community health. It also conducts professional education programs in these same areas, including the sponsorship of medical symposia and conferences, and the publication of scientific journals. The Association also provides grants for research and for the training of researchers.

American Lung Association National Office and its constituent and affiliate Lung Associations are tax-exempt organizations under Section 501(c)(3) of the Internal Revenue Code.

NOTE 2 – SUMMARY OF SIGNIFICANT ACCOUNTING POLICIES

Principles of Consolidation – The financial statements include ALA, its 59 constituent Lung Associations, and 69 affiliate Lung Associations. The constituent Lung Associations and their affiliates operate through agreements with, and under the authority of, ALA.

All intercompany transactions and balances have been eliminated in the consolidation.

Awards and Grants – The Association makes awards and grants for research and professional education purposes. Recipients are required to meet certain qualifications and to provide accountability to the Association for funds dispersed. The liability and related expense for awards and grants are recognized at the time of notification to, and acceptance by, the recipient.

Inventory – Inventory of educational and fund-raising materials is stated at the lower of cost or market on the first-in, first-out method.

Fixed Assets – Fixed assets are capitalized at cost. Depreciation is provided on the straight-line method over the estimated useful lives of the assets.

Donated Materials and Services – A substantial number of volunteers have donated significant amounts of time to the Association. However, since no objective basis exists for recording and assigning values to these services, they are not reflected in the accompanying financial statements. Similarly, the value of space and time contributed by various media for the Association's educational and fund-raising announcements is not subject to control or measurement and has not been recorded.

NOTE 3 – FIXED ASSETS

Land	$ 6,990,978
Buildings and building improvements	22,450,513
Leasehold improvements	489,174
Furniture, fixtures, and equipment	15,351,934
Automobiles	858,658
TOTAL	46,141,257
LESS ACCUMULATED DEPRECIATION AND AMORTIZATION	19,145,126
	$26,996,131

NOTE 4 – INVESTMENTS

	UNRESTRICTED FUND		DONOR RESTRICTED FUNDS		ENDOWMENT FUND		TOTAL	
	COST	MARKET	COST	MARKET	COST	MARKET	COST	MARKET
Equities	$10,098,394	$14,771,187	$ 47,973	$ 70,338	$ 6,428,279	$ 9,060,024	$16,574,646	$23,901,549
Bonds – corporate	4,887,397	5,105,455	32,820	32,878	2,267,848	2,383,208	7,188,065	7,521,541
U.S. government securities	12,553,586	13,006,656	399,390	409,895	2,887,157	3,089,771	15,840,133	16,506,322
Certificates of deposit	15,348,403	15,348,403					15,348,403	15,348,403
Mutual funds	5,117,236	5,324,096	69,334	69,334	45,113	43,107	5,231,683	5,436,537
Other	5,662,665	5,719,379	21,313	21,313	197,208	209,940	5,881,186	5,950,632
TOTAL	$53,667,681	$59,275,176	$570,830	$603,758	$11,825,605	$14,786,050	$66,064,116	$74,664,984

The net unrealized gain on investments for the year ended June 30, 1992 was $1,986,383.

NOTE 5 – LONG-TERM DEBT

Notes payable in installments through 1997 at a weighted average interest rate of 10.2% at June 30, 1992	$ 584,277
Mortgages payable in installments through 2010 at a weighted average interest rate of 9.1% at June 30, 1992	3,471,622
Capitalized lease obligations payable in installments through 1997 at a weighted average interest rate of 12.1% at June 30, 1992	247,273
TOTAL DEBT	4,303,172
Less current maturities	1,221,391
TOTAL LONG-TERM DEBT	$3,081,781

Long-term debt maturing during each of the next five years is as follows:

1992–93	$1,221,391
1993–94	242,078
1994–95	434,227
1995–96	161,409
1996–97	504,140

Interest expense for the year ended June 30, 1992 was $406,675.

NOTE 6 – LEASE COMMITMENTS

At June 30, 1992, the future minimum rental commitments for all noncancelable operating leases expiring at various dates are as follows:

1992–93	$3,117,358
1993–94	2,820,745
1994–95	2,269,736
1995–96	1,912,458
1996–97	1,654,243

Rent expense for the year ended June 30, 1992 was $3,546,114.

NOTE 7 – RETIREMENT PLANS

The Association has a defined benefit pension plan maintained by 96 Lung Associations participating on a noncontributory basis and 13 participating with contributory requirements. In addition to this plan, two Lung Associations have other, noncontributory defined benefit plans. The pension plans cover substantially all employees in the participating Lung Associations.

The following table sets forth the plan's funded status and amounts recognized in the Association's financial statements as of June 30, 1992:

Actuarial present value of benefit obligations:	
Accumulated benefit obligation, including vested benefits of $20,943,589	$21,480,474
Projected benefit obligation for service rendered to date	$(32,352,793)
Plan assets at fair value (primarily the general assets of Metropolitan Life Insurance and affiliated companies and listed securities)	37,365,842
Plan assets in excess of projected benefit obligation	$5,013,549
Unrecognized net gain from past experience different from that assumed and effects of changes in assumptions	770,246
Unrecognized prior service cost	418,688
Unrecognized net obligation at transition	(4,647,532)
PREPAID PENSION COST INCLUDED IN BALANCE SHEET	$1,554,951

Net periodic pension cost for 1991-92 includes the following components:

Service cost of benefits earned during the period	$2,185,487
Interest cost on projected benefit obligation	2,354,261
Actual return on plan assets	(2,871,817)
Net amortization and deferral	(781,973)
NET PENSION EXPENSE	$ 885,958

The weighted-average discount rate and rate of increase in future compensation levels used in determining the actuarial present value of the projected benefit obligation were 8.5 percent and 5 percent, respectively. The expected long-term rate of return on assets was 9 percent.

Certain associations participate in defined contribution retirement plans. Pension expense for these plans for the year ended June 30, 1992 was $195,944.

NOTE 8 – ALLOCATION OF JOINT COSTS

During the year ended June 30, 1992, the Association incurred joint costs of $26,191,153 for informational materials and activities that included fund-raising appeals. Of these costs, $12,481,602 were allocated to fund raising, $1,931,978 to environmental health, $3,852,563 to smoking education, $4,449,324 to lung disease, $3,138,034 to community health services, and $337,652 to management and general.

NOTE 9 – PRIOR PERIOD ADJUSTMENTS

Subsequent to the issuance of its 1991 financial statements, ALA discovered errors in the computation of Unrelated Business Income Tax (UBIT) and the classification of restricted expenses. In addition, ALA changed its method of depreciation from one that was not in accordance with generally accepted accounting principles to one that is. This type of change in accounting principle and the previously mentioned errors require retroactive adjustment and restatement. Accordingly, the fund balances at June 30, 1991 have been decreased by $597,736. Of that amount, $263,903 is applicable to 1991 and decreased that year's surplus from $1,560,374 (previously reported) to $1,296,471. The remaining $333,833 is applicable to years prior to 1991, and has been charged to fund balances at July 1, 1990.

NOTE 10 – REVOCABLE & IRREVOCABLE TRUSTS (UNAUDITED)

The American Lung Association of Los Angeles County ("ALALAC"), one of the ALA affiliate Lung Associations, reported to ALA that it acts as trustee, or has been named as successor trustee, for various revocable and irrevocable trusts. The assets of such trusts are not assets of the affiliate and therefore, are not recorded in the financial statements of the affiliate or of the Association.

The affiliate has reported on an unaudited basis that, as of June 30, 1992, it was a beneficiary under revocable and irrevocable trust agreements the principal amounts of which were valued at $28,181,000 and $3,267,000, respectively, and in which the affiliate's beneficial interests were valued in the amounts of $14,310,000 and $2,447,000, respectively. ALALAC has been advised by ALA of the need to submit audited results with respect to the trusts, and ALALAC has retained independent certified public accountants to conduct an audit of such trusts and such an audit is in progress as of March 1, 1993.

ALALAC, acting in its capacity as trustee, is not currently covered by fiduciary liability insurance. The absence of such insurance coverage does not mean that an asset has been impaired or a liability has been incurred by ALALAC. However, should an asset have been impaired or a liability been incurred, an estimate of the possible loss or range of loss is not presently determinable. The financial information reported to ALA indicates that ALALAC's unrestricted net assets were limited to $5.2 million as of June 30, 1992.

NOTE 11 – COMPARATIVE TOTALS

The amounts shown for the year ended June 30, 1991 in the accompanying financial statements are included to provide a basis for comparison with 1992 and present summarized totals only. Accordingly, the 1991 totals are not intended to present all information necessary for a fair presentation in conformity with generally accepted accounting principles.

Certain prior year's amounts have been reclassified to conform to the current year's presentation.

LOEB & TROPER
Certified Public Accountants
270 Madison Avenue, New York, NY 10016

BOARD OF DIRECTORS AND COUNCIL
AMERICAN LUNG ASSOCIATION

We have audited the accompanying balance sheet of American Lung Association National Office as of June 30, 1992, and the related statements of support, revenue, expenses and changes in fund balances, and functional expenses for the year then ended. These financial statements are the responsibility of the Association's management. Our responsibility is to express an opinion on these financial statements based on our audit.

We conducted our audit in accordance with generally accepted auditing standards. Those standards require that we plan and perform the audit to obtain reasonable assurance about whether the financial statements are free of material misstatement. An audit includes examining, on a test basis, evidence supporting the amounts and disclosures in the financial statements. An audit also includes assessing the accounting principles used and significant estimates made by management, as well as evaluating the overall financial statement presentation. We believe that our audit provides a reasonable basis for our opinion.

In our opinion, the financial statements referred to above present fairly, in all material respects, the financial position of American Lung Association National Office as of June 30, 1992, and the results of its operations for the year then ended in conformity with generally accepted accounting principles.

Loeb & Troper

CERTIFIED PUBLIC ACCOUNTANTS
September 30, 1992, except for Note 9 as to which the date is November 23, 1992.

American Lung Association National Office

BALANCE SHEET JUNE 30, 1992 (With comparative totals for 1991)

	UNRESTRICTED							TOTAL	
ASSETS	OPERATING FUND	BOARD DESIGNATED	TOTAL	DONOR RESTRICTED	EQUIPMENT FUND	ENDOWMENT FUND	ANNUITY GIFT FUND	1992	1991 (NOTE 9)
Current assets									
Cash	$ 17,826		$ 17,826				$ 9,718	$ 27,544	$ 161,357
Short-term investments	5,226,659	$ 86,521	5,313,180	$ 303,348		$ 31,915	538,528	6,186,971	3,214,362
Receivables – primarily from constituent and affiliate associations (net of allowance for doubtful accounts of $192,931 in 1992 and $79,088 in 1991)	3,975,140	46,543	4,021,683	841,788				4,863,471	4,381,806
Pledges receivable				252,500				252,500	378,750
Inventory of educational and fund-raising materials	2,930,445		2,930,445					2,930,445	2,631,170
Prepaid expenses and other current assets (Note 5)	322,565		322,565	17,754				340,319	612,982
TOTAL CURRENT ASSETS	12,472,635	133,064	12,605,699	1,415,390		31,915	548,246	14,601,250	11,380,427
Noncurrent assets									
Pledges receivable				185,500				185,500	252,500
Fixed assets (net of accumulated depreciation and amortization of $1,421,181 in 1992 and $1,024,346 in 1991) (Note 7)					$1,763,419			1,763,419	1,687,642
Investments – at cost (market value $8,843,458 in 1992 and $12,861,304 in 1991) (Note 3)	5,614,979		5,614,979	11,700		690,952		6,317,631	10,598,193
TOTAL NONCURRENT ASSETS	5,614,979		5,614,979	197,200	1,763,419	690,952		8,266,550	12,538,335
TOTAL ASSETS	$18,087,614	$133,064	$18,220,678	$1,612,590	$1,763,419	$722,867	$548,246	$22,867,800	$23,918,762
LIABILITIES AND FUND BALANCES									
Current liabilities									
Accounts payable and other current liabilities	$ 3,921,275	$ 6,000	$ 3,927,275	$ 2,690	$ 45,535			$ 3,975,500	$ 4,736,590
Awards and grants payable (Note 2)	1,250,521		1,250,521	224,500				1,475,021	3,146,837
Deferred revenue	2,155,833		2,155,833	1,288,685				3,444,518	2,366,806
Due to constituent and affiliate associations	720,813		720,813					720,813	300,000
TOTAL CURRENT LIABILITIES	8,048,442	6,000	8,054,442	1,515,875	45,535			9,615,852	10,550,233
Noncurrent liabilities									
Deferred revenue	867,989		867,989	73,000			$ 45,069	986,058	1,016,900
Due to constituent and affiliate associations							503,177	503,177	504,764
Other noncurrent liabilities					73,012			73,012	118,547
TOTAL NONCURRENT LIABILITIES	867,989		867,989	73,000	73,012		548,246	1,562,247	1,730,391
TOTAL LIABILITIES	8,916,431	6,000	8,922,431	1,588,875	118,547		548,246	11,178,099	12,270,524
FUND BALANCES	9,171,183	127,064	9,298,247	23,715	1,644,872	722,867		11,689,701	11,648,238
TOTAL LIABILITIES AND FUND BALANCES	$18,087,614	$133,064	$18,220,678	$1,612,590	$1,763,419	$722,867	$548,246	$22,867,800	$23,918,762

See independent auditor's report
The accompanying notes are an integral part of these statements.

American Lung Association National Office

STATEMENT OF SUPPORT, REVENUE, EXPENSES AND CHANGES IN FUND BALANCES YEAR ENDED JUNE 30, 1992 (With comparative totals for 1991)

	UNRESTRICTED						TOTAL	
PUBLIC SUPPORT AND REVENUE	OPERATING FUND	BOARD DESIGNATED	TOTAL	DONOR RESTRICTED	EQUIPMENT FUND	ENDOWMENT FUND	1992	1991 (NOTE 9)
Public support								
Shareable income from constituent associations	$ 8,886,707		$ 8,886,707				$ 8,886,707	$ 9,010,471
Nonshareable public support	184,413		184,413	$ 717,402		$(250,000)	651,815	1,394,622
Bequests	59,042		59,042	5,239		1,000	65,281	115,215
Special events (net of direct expenses of $44,911 in 1992 and $45,330 in 1991)				230,966			230,966	216,845
TOTAL PUBLIC SUPPORT	9,130,162		9,130,162	953,607		(249,000)	9,834,769	10,737,153
GRANTS FROM GOVERNMENT AGENCIES				698,671			698,671	475,325
Revenue								
Net results of billings for supplies and services to constituent and affiliate associations (net of related expenses of $20,759,003 in 1992 and $17,555,479 in 1991)	328,796		328,796				328,796	396,926
Other sales – program outreach	3,717,501		3,717,501				3,717,501	
Investment income	594,916		594,916	46,283			641,199	857,339
Gain on sale of investments	493,820		493,820			22,230	516,050	184,600
Scientific journals	2,700,417		2,700,417				2,700,417	2,273,809
Membership dues	1,113,765		1,113,765				1,113,765	794,392
International Conference	2,391,174		2,391,174				2,391,174	1,748,945
Program service fees, royalties and other	1,120,604	$113,230	1,241,834	48,810			1,290,644	730,808
TOTAL REVENUE	12,460,993	113,230	12,582,223	95,093		22,230	12,699,546	6,986,819
TOTAL PUBLIC SUPPORT AND REVENUE	21,591,155	113,230	21,712,385	1,747,371		(226,770)	23,232,986	18,199,297
EXPENSES								
Program services								
Environmental health	2,663,732		2,663,732	786,755	53,410		3,503,897	2,825,792
Smoking education	3,676,735		3,676,735	61,575	23,850		3,762,160	1,050,673
Lung disease	7,512,094		7,512,094	1,041,240	87,070		8,640,404	7,743,859
Community health services	3,970,516	89,686	4,060,202	22,932	104,511		4,187,645	3,856,737
TOTAL PROGRAM SERVICES	17,823,077	89,686	17,912,763	1,912,502	268,841		20,094,106	15,477,061
Supporting services								
Management and general	2,149,951		2,149,951		105,875		2,255,826	2,637,598
Fund raising	819,472		819,472		22,119		841,591	721,592
TOTAL SUPPORTING SERVICES	2,969,423		2,969,423		127,994		3,097,417	3,359,190
TOTAL EXPENSES	20,792,500	89,686	20,882,186	1,912,502	396,835		23,191,523	18,836,251
Excess (deficiency) of public support and revenue over expenses before loss on Trek	806,655	23,544	830,199	(165,131)	(396,835)	(226,770)	41,463	(636,954)
Loss on Trek								(75,673)
Excess (deficiency) of public support and revenue over expenses	806,655	23,544	830,199	(165,131)	(396,835)	(226,770)	41,463	(712,627)
Other changes in fund balances								
Fixed asset acquisitions from unrestricted fund	(460,046)		(460,046)		460,046			
Amortization of capital lease obligation	(31,750)		(31,750)		31,750			
Fund balances – beginning of year – as originally stated	8,742,887	103,520	8,846,407	601,305	1,848,625	949,637	12,245,974	12,694,698
Prior period adjustments (Note 9)	113,437		113,437	(412,459)	(298,714)		(597,736)	(333,833)
Fund balances – beginning of year – restated	8,856,324	103,520	8,959,844	188,846	1,549,911	949,637	11,648,238	12,360,865
FUND BALANCES – END OF YEAR	$ 9,171,183	$127,064	$ 9,298,247	$ 23,715	$1,644,872	$722,867	$11,689,701	$11,648,238

See independent auditor's report

The accompanying notes are an integral part of these statements.

American Lung Association National Office

STATEMENT OF FUNCTIONAL EXPENSES YEAR ENDED JUNE 30, 1992 (With comparative totals for 1991)

	PROGRAM SERVICES				
	ENVIRONMENTAL HEALTH	SMOKING EDUCATION	LUNG DISEASE	COMMUNITY HEALTH SERVICES	TOTAL
Salaries (Note 8)	$ 961,590	$ 412,138	$1,533,653	$1,787,047	$ 4,694,428
Employee health and retirement benefits	100,249	46,943	161,447	194,640	503,279
Payroll taxes	72,889	32,548	118,824	142,646	366,907
TOTAL SALARIES AND RELATED EXPENSES	1,134,728	491,629	1,813,924	2,124,333	5,564,614
Professional fees	590,607	72,771	648,575	320,546	1,632,499
Occupancy	247,544	87,188	346,353	410,566	1,091,651
Printing, publications, and audiovisual materials	624,977	2,839,827	1,413,998	240,250	5,119,052
Media expenses	16,401	8,447	8,587	29,295	62,730
Office and cleaning supplies	30,956	9,097	40,187	42,172	121,512
Equipment rental and maintenance	61,717	9,018	117,568	46,552	234,855
Postage and shipping	80,202	13,460	151,916	102,989	348,567
Telephone	35,496	11,706	50,824	67,715	165,741
Meetings and conferences					
Staff	9,832	1,905	16,455	69,537	97,729
International Conference	83,909	374	191,731	63,393	339,407
Board and professional committees	177,797	15,439	401,178	175,327	769,741
Travel and local transportation	49,649	26,498	71,511	179,779	327,437
Awards and grants			2,981,511	19,351	3,000,862
Dues and subscriptions	12,236	2,368	5,819	20,829	41,252
Insurance	8,207	3,566	13,537	15,837	41,147
Support of other organizations	16,500	130,890	213,395	36,444	397,229
Support of other Lung Associations	262,416	12,000	46,000	108,382	428,798
Miscellaneous	8,213	2,127	20,265	9,837	40,442
TOTAL EXPENSES BEFORE DEPRECIATION AND AMORTIZATION	3,450,487	3,738,310	8,553,334	4,083,134	19,825,265
Depreciation and amortization	53,410	23,850	87,070	104,511	268,841
TOTAL EXPENSES	$3,503,897	$3,762,160	$8,640,404	$4,187,645	$20,094,106

See independent auditor's report. The accompanying notes are an integral part of these statements.

AMERICAN LUNG ASSOCIATION (NATIONAL OFFICE)

NOTES TO FINANCIAL STATEMENTS JUNE 30, 1992

NOTE 1 – DESCRIPTION OF ORGANIZATION

American Lung Association National Office (ALA) is a national not-for-profit voluntary health organization dedicated to the fight against lung disease. ALA fights lung disease through programs of public education on environmental health, smoking, lung disease, and community health. It also conducts professional education programs in these same areas, including the sponsorship of medical symposia and conferences, and the publication of scientific journals. ALA also provides grants for research and for the training of researchers.

ALA is a tax-exempt organization under Section 501(c)(3) of the Internal Revenue Code.

NOTE 2 – SUMMARY OF SIGNIFICANT ACCOUNTING POLICIES

Awards and Grants – ALA makes awards and grants for research and professional education purposes equal to one-third of the shareable revenue, less 15 percent for administration, received from its constituents and affiliates during the year. Recipients are required to meet certain qualifications and to provide accountability to ALA for funds disbursed. The liability and related expense for awards and grants are recognized at the time of notification to, and acceptance by, the recipient.

In prior years, payment of awards occurred in the year after the liability was recognized. In an attempt to accelerate the availability of monies to awardees, one-half of the 1992 awards were paid in June, 1992, resulting in a decrease in the current year's awards and grants liability of approximately $1,475,000.

Inventory – Inventory of educational and fund-raising materials is stated at the lower of cost or market on the first-in, first-out method.

Donated Materials and Services – A substantial number of volunteers have donated significant amounts of time to ALA. However, since no objective basis exists for recording and assigning values to these services, they are not reflected in the accompanying financial statements. Similarly, the value of space and time contributed by various media for ALA educational and fund-raising announcements is not subject to control or measurement and is not recorded.

Additional accounting policies are included in other footnotes.

NOTE 4 – LEASES

ALA leases office space under operating leases at various locations throughout the United States. The annual rentals are subject to escalation agreements and periodic rate increases. Rent expense for the year ended June 30, 1992 was approximately $1,767,000. This includes rent expense relating to the regional fund-raising centers and supply service which are netted into the supply service operations. ALA has a capital lease agreement for its computer hardware. The minimum annual rental payments for noncancellable leases are as follows.

	OPERATING LEASES	CAPITAL LEASES
1992–93	$1,491,535	$ 45,535
1993–94	1,429,868	47,066
1994–95	1,385,429	25,946
1995–96	1,259,514	
1996–97	1,293,351	

NOTE 3 – INVESTMENTS

	UNRESTRICTED FUND		DONOR RESTRICTED FUNDS		ENDOWMENT FUND		TOTAL	
	COST	MARKET	COST	MARKET	COST	MARKET	COST	MARKET
U.S. Treasury securities	$ 915,448	$ 960,343			$198,419	$210,781	$1,113,867	$1,171,124
Equities	3,097,002	5,199,113	$11,700	$18,779	391,356	628,196	3,500,058	5,846,088
Government agency bonds	1,403,354	1,508,808			101,177	107,438	1,504,531	1,616,246
Foreign bonds	199,175	210,000					199,175	210,000
	$5,614,979	$7,878,264	$11,700	$18,779	$690,952	$946,415	$6,317,631	$8,843,458

SUPPORTING SERVICES			TOTAL	
MANAGEMENT AND GENERAL	FUND RAISING	TOTAL	1992	1991 (NOTE 9)
$ 940,339	$380,617	$1,320,956	$ 6,015,384	$ 5,546,098
122,523	41,190	163,713	666,992	605,787
80,368	30,166	110,534	477,441	444,580
1,143,230	451,973	1,595,203	7,159,817	6,596,465
413,048	144,656	557,704	2,190,203	2,078,361
196,323	73,687	270,010	1,361,661	1,115,180
24,091	5,887	29,978	5,149,030	2,254,406
			62,730	33,163
25,906	10,028	35,934	157,446	117,225
42,712	7,304	50,016	284,871	320,660
35,651	13,192	48,843	397,410	344,415
28,937	8,959	37,896	203,637	195,134
19,356	5,447	24,803	122,532	134,679
			339,407	268,741
123,602	41,179	164,781	934,522	817,851
33,184	33,674	66,858	394,295	373,210
			3,000,862	3,059,378
5,340	1,619	6,959	48,211	37,515
8,801	3,403	12,204	53,351	45,524
30,000	17,250	47,250	644,479	309,730
			428,798	308,990
19,770	1,214	20,984	61,426	72,067
2,149,951	819,472	2,969,423	22,794,608	18,432,582
105,875	72,119	127,994	396,835	403,749
$2,255,826	$841,591	$3,097,417	$23,191,523	$18,836,251

NOTE 5 – RETIREMENT PLAN

ALA has a noncontributory defined benefit pension plan covering substantially all of its employees.

The following table sets forth the plan's financial status and amounts recognized in ALA's financial statements as of June 30, 1992:

Actuarial present value of benefit obligations:	
Accumulated benefit obligation, including vested benefits of $3,456,886	$(3,576,672)
Projected benefit obligation for service rendered to date	$(5,153,906)
Plan assets at fair value (primarily the general assets of Metropolitan Life Insurance and affiliated companies and listed securities)	5,335,094
Plan assets in excess of projected benefit obligation	181,188
Unrecognized net gain from past experience different from that assumed and effects of changes in assumptions	590,675
Unrecognized prior service cost	63,023
Unrecognized net obligation at transition	(690,205)
Prepaid pension cost included in balance sheet	$ 144,681
Net pension expense for 1991-92 includes the following components:	
Service cost of benefits earned during the period	$366,594
Interest cost on projected benefit obligation	369,475
Actual return on plan assets	(430,208)
Net amortization and deferral	(86,474)
NET PENSION EXPENSE	$219,387

The weighted-average discount rate and rate of increase in future compensation levels used in determining the actuarial present value of the projected benefit obligation were 8.5 percent and 5 percent, respectively. The expected long-term rate of return on assets was 9 percent.

NOTE 6 – RELATED PARTIES

At June 30, 1992, there were 59 constituent Lung Associations, which have jurisdiction over specific geographic areas and/or one or more of 69 affiliate Lung Associations. Each constituent and/or affiliate Lung Association is required to remit 10 percent of its shareable revenue to ALA, one-third of which, less a 15 percent administrative charge, is used to fund research. ALA also provides national leadership and certain services to its constituent and affiliate Lung Associations.

ALA provides certain supplies to constituent and affiliate Lung Associations, including Christmas Seals® and other fund-raising and health education materials, at a price which approximates cost. ALA also operates regional fund-raising centers, the costs of which are passed on directly to the participating constituent and affiliate Lung Associations. Revenue from supplies and services provided to constituent and affiliate Lung Associations is reflected net of related costs. For the year ended June 30, 1992, this revenue is also reflected net of a rebate to constituent and affiliate Lung Associations of $285,000.

NOTE 7 – FIXED ASSETS

Fixed assets are capitalized at cost. Depreciation is provided using the straight-line method over the estimated useful lives of the assets (equipment and capital lease equipment for five years, furniture and fixtures for 10 years, and leasehold improvements over the remaining term of the lease). In the beginning of 1992, the useful lives of several asset categories were revised to reflect the period of benefit or service to ALA. This is considered a change in estimate which was recorded on a prospective basis.

	COST	ACCUMULATED DEPRECIATION AND AMORTIZATION	NET
Leasehold improvements	$ 184,881	$ 47,006	$ 137,875
Equipment	1,666,902	987,954	678,948
Capital lease equipment	192,857	93,804	99,053
Furniture and fixtures	1,139,960	292,417	847,543
	$3,184,600	$1,421,181	$1,763,419

NOTE 8 – VACATION ACCRUAL

Effective July 1, 1991, the policy relating to payment of accrued vacation to terminated employees was revised. Consequently, salary expense increased approximately $240,000 for the year ended June 30, 1992.

NOTE 9 – PRIOR PERIOD ADJUSTMENTS

Subsequent to the issuance of its 1991 financial statements, ALA discovered errors in the computation of Unrelated Business Income Tax (UBIT) and the classification of restricted expenses. In addition, ALA changed its method of depreciation from one that was not in accordance with generally accepted accounting principles to one that is. This type of change in accounting principle and the previously mentioned errors require retroactive adjustment and restatement. Accordingly, the fund balance at June 30, 1991 has been decreased by $597,736. Of that amount, $263,903 is applicable to 1991 and increased that year's net loss from $448,724 (previously reported) to $712,627. The remaining $333,833 is applicable to years prior to 1991, and has been charged to the fund balance at July 1, 1990.

The underpaid UBIT, including interest and penalties, on advertising revenue from the scientific journals, totals $299,022. Of this amount, $62,928 relates to 1991 and has been reflected as a decrease in the revenue of the scientific journals and an increase in accounts payable for that year. The remaining $236,094 is applicable to years prior to 1991 and has been charged to the fund balance at July 1, 1990.

The depreciation adjustment totals $298,714. Of this amount, $200,975 relates to 1991 and has been reflected as an increase in depreciation expense and accumulated depreciation for that year. The remaining $97,739 is applicable to years prior to 1991 and has been charged to the fund balance at July 1, 1990.

Prior years' restricted expenses totaling $412,459 were incorrectly charged to the unrestricted fund. Of this amount, $246,845 relates to 1991 and was transferred from the restricted fund balance to the unrestricted fund balance at June 30, 1991. The remaining $165,614 relates to years prior to 1991 and has been transferred from the restricted fund balance to the unrestricted fund balance at July 1, 1990. The transfer between funds has no effect on 1991 comparative figures since 1991 fund detail is not presented herein.

NOTE 10 – COMPARATIVE TOTALS

The amounts shown for the year ended June 30, 1991 in the accompanying financial statements are included to provide a basis for comparison with 1992 and present summarized totals only. Accordingly, the 1991 totals are not intended to present all information necessary for a fair presentation in conformity with generally accepted accounting principles.

Certain prior year's amounts have been reclassified to conform to the current year's presentation.

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Testimony Before the Subcommittee on Public Buildings and Grounds,
US House of Representatives, Committee on Public Works and Transportation

By: J.G. Robertson, President
Healthy Buildings International, Inc. (HBI)
10378 Democracy Lane, Fairfax, VA 22030

April 22, 1993

Thank you for the opportunity to present testimony to you today concerning H.R. 881, a bill to restrict smoking in federal buildings. I am following up on the presentation given to you by one of my staff, Mr. Simon Turner. I understand that Mr. Turner described to you the widespread problems of Sick Building Syndrome (SBS) in office buildings in this country. Mr. Turner drew on our experience of having studied approximately 1,000 major buildings and outlined to you the concept of a Building Systems Approach to deal with all types of indoor pollutants. Inherent in Mr. Turner's presentation was the fact that no company that deals with indoor air quality issues can expect to reduce all pollutant levels to zero values.

Indeed, in virtually every environment there exist small quantities of pollutants. Office air, even so-called fresh outdoor air, contains numerous chemicals, including substances categorized by EPA as Group A carcinogens. In urban areas, the air inevitably contains quantifiable levels of numerous chemicals including the Group A carcinogens asbestos, benzene and others, plus it usually contains radon.

The objective therefore is not to aim for the unobtainable. It is to use good operating practices and, where appropriate, source management to maintain the air in a generally acceptable condition that ensures the comfort and well-being of the occupants. Today I plan to spend some time exploring in more depth the options available for federal facility managers to improve the air quality for all building occupants and productively accommodate smoking staff within their buildings. As the same time they must accommodate the requirements of the nonsmokers who have a right not to be exposed to any excessive amounts of other's smoke.

Before I proceed further, I wish to state that my company makes many presentations to such recognized associations as the American Institute of Architects, American Society of Heating, Refrigerating and Air Conditioning Engineers (ASHRAE), the Association of Energy Managers, the Gas Council, the Air Balance Council, the Building Owners and Managers Association (BOMA), and numerous Fortune 500 Corporations. Without exception, these groups complement us on sharing the results of a decade of research in this way. If, however, the tobacco industry asked us to present an identical presentation, we are likely to be branded as apologists to that industry; we are not. We are a dedicated team of consultants who believes in describing conditions exactly as we find them. Today I appear before you at the request and expense of the Tobacco Institute. However, I assure you that my findings, statements and recommendations are based on our independent research. Indeed, I challenge anyone to debate these issues with me and for anyone to point out where my statements depart from basic common sense.

Smoking Policy Options

Smoking restrictions in buildings have been part of American life over the past few years. But only recently have we seen a move to eliminate smoking entirely from office buildings. This has led to the somewhat pathetic sight of groups of smokers huddled in front of entrances of major buildings in the central business districts of many major US cities. Many of our property management and employer clients have been distinctly unhappy with this arrangement. They worry about:

- Productivity - Smokers are walking long distances to take a smoke break, staying away from their work stations longer than if they had somewhere to smoke on their own floor.

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- Security - It is dangerous for women especially to be hanging around on city streets, especially after dark, in many of our major cities. Bear in mind that they are not there exactly of their own volition.

- Public Image - Many employers and facility managers find the sight of individuals loitering around the front entrance of their properties to be inconsistent with the image of a productive and efficient company. Indeed, foreign visitors to one major corporation sympathized with management that they must have severe industrial relations problems in view of the picket line outside the entrances!

- Litter - The pavement outside the front entrances of most buildings is not equipped with facilities for smokers, which adds to the public image problem above.

- Also, especially when dealing with large numbers of the public (e.g. airports), groups of smokers outside entrances have to be negotiated by building users.

Thus we have been asked by a wide range of clients, from major corporations to hospitals, to help them accommodate smokers inside their buildings. This is consistent with the stated desire of this committee to be anti-smoking, but not anti-smoker. There are several progressive steps that can be used to accommodate different degrees of separation between the smoker and nonsmoker.

The first directive is that in all areas where national or local governments have enacted laws and regulations prohibiting and/or restricting smoking, then such laws should be obeyed. Also, smoking should be prohibited near flammable materials, in areas where employees handle hazardous substances, in food preparation areas, and in close proximity to sensitive machinery or equipment. In all other areas, a common-sense approach to the issue of smoking policies is of paramount importance.

Since we are dealing with a wide variety of individuals working in a multiplicity of different buildings, it is inevitable that there is no one formula that satisfies all people at all times. We suggest therefore the introduction of a step-wise series of options moving from the current practice to an informal policy of accommodation through progressively more restrictive options.

Status Quo

In buildings that have adequate ventilation and those experiencing no significant complaints of air quality issues including tobacco smoke — why change? Discretionary smoking is practiced in many buildings. In such buildings that meet or exceed the ventilation rates of ASHRAE 62-1989, complaints are rare.

Many buildings using ASHRAE's minimum ventilating standards, with or without dedicated smoking areas, practice acceptable air quality procedures. Return air from discretionary smoking areas, or designated smoking areas when ducted to the HVAC systems, can be filtered and diluted to such an extent that it meets the ASHRAE 62-1989 definition of acceptable outdoor air. Since this standard is based on the National Primary Ambient Air Quality Standards for outdoor air as set by the U.S. EPA, it is totally unreasonable that such supply air to the building could be judged to be

unacceptable. In essence, if the filtration and dilution process provides supply air quality equivalent to the outdoors — so-called fresh air — then no further refinement should be judged necessary.

Designated Smoking Areas

With some thoughtfulness in the selection of the smoking area with respect to prevailing ventilation conditions, the policy of designated smoking areas works very satisfactorily.

Physical grouping of smokers and nonsmokers in discrete areas frequently obviates the concern of sensitive nonsmokers. In doing so, it is important to utilize the prevailing air currents and routes of supply and exhaust of the air. Whenever feasible, the smoking area should be concentrated nearer the exhaust grilles with the nonsmokers closer to the supply grilles. Providing the exhaust systems are sized properly, such an arrangement will ensure that the air movements will be diverted from nonsmoking areas to smoking areas and then to room exhaust. This minimizes the migration of tobacco smoke from smoking to nonsmoking areas. Thereafter, the exhaust air should exit the building or, provided it is filtered and diluted to meet nationally agreed outdoor air quality standards, it can be recycled back to the air supply system.

Air Cleaning Equipment

In designated smoking areas, supplementary air cleaning offers some worthwhile advantages. Equipment marketed specifically for removal of tobacco smoke components from room air include electrostatic precipitators, activated charcoal filters and high efficiency filters such as HEPA filtration units. All three have their place in removing substances from the air. The electrostatic precipitators and HEPA filters are very efficient at removing the finer so-called respirable suspended particulates (RSP) from the air. Correctly sized, well-serviced electrostatic precipitators or HEPA filters can remove 90 to 98 percent of these airborne particles. Neither of these filters, however, can remove gaseous components. Here we need the use of adsorbent filters using activated charcoal, potassium permanganate impregnated aluminum oxide pellets or other proprietary adsorbents. These, again presupposing they are sized correctly and serviced regularly, can remove substantial amounts of unpleasant odors, gases, nicotine, etc.

The most effective device for removing perceived signs (olfactory or visual) of tobacco smoke appears to be a combination of a "roughing" filter followed by an electrostatic precipitator or HEPA unit, followed by an adsorbent filter. Several proprietary designs of this combination are readily available worldwide. The combination of separate smoking and nonsmoking areas coupled to the use of supplementary filtration in the smoking sections and presupposing adequate ventilation rates, i.e., ASHRAE 62-1989 Standard rates, is usually more than adequate to reasonably satisfy both smokers and nonsmokers.

ASHRAE 62-1989 — Design of Smoking Lounge

Dedicated smoking lounges can be set up in most buildings. The specification of a separate ventilating system is accomplished by providing the designated lounge with a local exhaust ventilating system.

ASHRAE's suggested ventilation rate for a dedicated smoking lounge is 60 cfm/person based on an occupancy rate of approximately 15 square feet per smoker. This equates to 4 cfm of exhaust air per square foot of lounge area. In sizing a dedicated smoking lounge, multiply the area of the lounge in square feet by four to yield the required capacity of the exhaust fan in cubic feet per minute.

In retrofitting an existing office or store room to function as a smoking lounge, the following steps are suggested:

- Disconnect and seal off any existing return air grilles;
- Install, preferably in a central location of the ceiling, a designated exhaust grille connected to an exhaust fan rated as described above;
- The discharge from the exhaust system should be ducted to any dedicated exhaust system serving the building, e.g., kitchen or toilet exhausts. Alternatively, the discharge can be ducted to any existing exhaust shafts or when feasible, directly through the wall to the exterior of the building. Most authorities permit linking dedicated exhaust systems together in a common duct, but individual building owners should check their local authority for regulations to avoid contravening local building codes.
- In the case of heavy utilization of the smoking lounge, it would be prudent to install in the lounge supplementary filtration. High efficiency media filters or electrostatic precipitators are recommended. The capacity of such room filters should be selected to ensure that the total volume of room air is filtered every five minutes.

$$\textit{i.e. Minimum cfm capacity of filter} = \frac{\textit{Volume of Room ft}^3}{5}$$

- In providing exhaust air capacity at a rate of 4 cfm/square foot, the smoking room will develop a negative air pressure with respect to adjacent areas. Thus, air flow will be from the adjacent areas into the smoking room, precluding the release of smoke into the adjacent areas. The movement of air from the higher pressure areas to the negative pressure areas is defined as transfer air. The use of transfer air for smoking lounges, toilets, etc. is recommended by ASHRAE to preclude the movement of annoying odors and other noxious substances into other occupied areas.

Displacement Ventilation Designs

Displacement ventilation occurs when the ventilation air flows past the contaminant source and sweeps the contaminant towards an exhaust. In the case of smoking rooms, the most effective route is to channel all the supply air from floor level and allow it to move vertically through the room, exhausting at ceiling height. This ensures that all the substances present in the indoor air, such as dusts, skin scale, tobacco smoke, body odors, microbes, exhaled breath, etc. will also rise vertically above the room occupants and be removed into the exhaust located in the ceiling. Since most of the emissions of pollutants from human activities are warm relative to the surrounding air, there is a

natural tendency for them to move upwards on thermal currents. The upward flow of ventilation air speeds up this process in displacement ventilation designs.

Two options for smoking room designs are suggested. One requires a raised floor air supply system similar to the classical computer room design, the other can be installed on any existing floor using specially designed low velocity air diffusers. These designs are referred to as the Filtered Air Control Technology (FACT) and the Halton DisplaceVent System.

Halton DisplaceVent System

This is classical displacement ventilation as commonly practiced throughout Scandinavia and Europe. The supply air to the designated room is conditioned to a temperature of 66 to 68°F. This air is then ducted usually from conventional ducts in the ceiling void via vertical ducts to floor-mounted specially designed low velocity diffusers.

In essence, the supply air, several degrees cooler than the room air, spills across the floor forming a lake of clean air. Immediately on entering the room, the air starts to be warmed and therefore rises, in classical thermal currents, towards the ceiling. Any heat sources in the room, including people, creates a "heat bridge" towards the ceiling. Indeed, the supply air is induced across the surface of the room occupants forming a plume above them moving to the ceiling. This means that clean air is continually drawn past the breathing zone of the room occupants. This concept is effectively a self-regulating form of demand ventilation. The more people present in the room, the faster the upward air flow due to increased thermal loads.

Exhaust is provided at a centrally located diffuser located in the ceiling. An advantage of this DisplaceVent technique is that it is easy to retrofit into any existing areas. The low velocity diffusers come in a variety of shapes to best accommodate the geography of the room concerned. Typically they are corner-mounted to minimize loss of usable floor space, but wall units can be located inside hollow walls when available.

These displacement ventilation techniques are many times more efficient than conventional mixing type ventilation where both supply and return air are delivered or removed from the space via a common ceiling. Indeed, throughout Europe and Scandinavia, many buildings including commercial offices are preferring such displacement ventilation techniques for all occupied spaces whether smoking is practiced or not.

FACT

The FACT system, designed by Philip Morris research engineers, provides a constant volume of supply air, moving at a rate of ten feet per minute through a perforated raised flooring system. This velocity of air moving uniformly upwards throughout the room displaces the room air in a piston or plug flow, vertically upwards. The displaced air carries with it all airborne substances, which are removed via the exhaust/return air system. Typically 50 percent of the air is exhausted, the other 50 percent constitutes return air. Outside air is mixed with the air from the ducted return and the mixture is scrubbed via high quality electronic air cleaners and activated charcoal-based gas phase filters. The clean air is then conditioned via conventional heating and cooling coils before being returned to the subfloor to start the process over again.

Tests conducted by independent air quality contractors prove that this system delivers air to the breathing zone of the room occupants at a quality substantially better than conventional nonsmoking offices, or the outdoor air.

Since the design is based on a raised access floor system, this design will not be practical in all buildings. However, when new construction or major remodelling is planned, this design is very much worthy of consideration.

Tobacco Smoke — An Object Lesson, A Litmus Test

Returning to the overall problem of indoor air pollution due to poor ventilation, perhaps a more serious issue for occupants of under-ventilated spaces is not so obvious. The spread of infectious diseases in poorly ventilated spaces is well documented. Radon — a colorless, odorless and extremely dangerous gas — can be concentrated in such an environment and concentrations of a wide range of volatile organic chemicals invariably increase as the ventilation rates decrease.

An interesting example of the need for optimum ventilation rates would be the hospital operating room. Not only are there increased risks of exposure to germs in this environment, but the widespread use of anesthetic gases demands high ventilation rates. Accordingly, it is not unusual for regular checks of ventilation effectiveness to be carried out in operating rooms. One technique involves the release of synthetic smoke. The visible smoke illustrates which air currents convey the air away from the room and indicates the efficiency of pollutant removal. If the smoke lingers, there is good cause for concern. Not only would the germs be trapped, leading to risks of cross infection, but also anesthetic gases could be trapped with potentially disastrous effects on the operating teams.

By analogy, the visible tobacco smoke in an office demonstrates the ventilating system capability. Excessive smoke accumulation is visible proof of deficiencies of the ventilation system. If such accumulations led to complaints in the past, sometimes the quick and easy fix was to ban smoking. However, unless the root cause of the problem was addressed — deficient ventilation capacity — then the quick fix cannot eliminate the more insidious pollutant problems that must still be present in a poorly ventilated area. In future, litigating attorneys may be quick to grasp the significance of the previous smoking bans. Low cost cosmetic improvements introduced to placate anti-smokers may buy short-term peace of mind. Unfortunately, failure to eliminate the real cause of the problems must eventually come home to roost when more chronic problems of sickness manifest themselves in the poorly ventilated office.

Indeed, the Environmental Protection Agency (EPA) headquarters building in Washington, DC illustrates just such a scenario. An inadequately ventilated building trapped all types of indoor pollutants. Early complaints of tobacco smoke accumulations led to smoking bans — much to the delight of many anti-smokers. Within two years of the smoking bans, numerous EPA employees have filed a multi-million dollar lawsuit against the managers of the building alleging toxic exposure to various volatile organic chemicals caused by inadequate ventilation.

It is unfortunate that those managers failed to recognize that the previously trapped smoke was "the canary in the coal mine." Even the anti-smokers amongst the plaintiffs who may now have developed irreversible long term injuries deserve sympathy simply because they failed to identify the true cause of their discomfort. Perhaps worst of all, from the building manager's point of view, is

the fact that the plaintiff attorneys now have hard evidence that the building was operating for some considerable time with inadequate ventilation — a system that could not handle smoke cannot handle dusts, bacteria, fungi and volatile organic chemicals.

The fact is that properly designed and correctly operated ventilating systems are capable of handling all indoor substances, including smoke. This further illustrates the necessity of addressing any indoor air quality issue with a Building Systems Approach. When complaints of smoking are first voiced, it is incumbent on the management to correct the long term problem — poor air quality — and then, only if still necessary, address the issue of smoking. Forward-thinking managers who see the complete picture achieve best results. Complaints need to be correctly understood. Invariably a professionally conducted environmental evaluation of the workplace will identify existing or potential problem areas; attention to faults identified usually result in dramatically better air quality; morale improves; productivity increases; and usually the precursor of the problem, the smoke accumulation, evaporates.

Optimization of the Workplace — Building Systems Approach

In the future it seems inevitable that some form of regulation will be required to control indoor air quality as a whole. In a 1987 report "Policies & Procedures for Control of Indoor Air Quality", the National Research Council (NRC) concluded that the levels of both chemical and biological contaminants were strongly associated with the cleanliness of the heating, ventilation and air-conditioning system. They also noted the complex nature of the indoor air and the difficulties of identifying sources of pollution and suggested that solutions can usually be implemented without such identification. The EPA itself in the "Research Needs Section (Volume III) of a report to Congress issued in ....1990 noted that "more can be done to reduce overall exposures and risks by altering building designs and ventilation patterns than by approaching the problem source-by-source or pollutant-by-pollutant".

With the aim of improving comfort, productivity and attendance, forward-thinking managers are finding it cost effective to optimize the workplace from an environmental perspective. The approach is applied to both existing and planned buildings in order to prevent problems rather than attempting to cure problems after the fact.

The cornerstone of this approach is the design, operation and maintainance of buildings to specified minimum environmental guidelines. Since the American Society of Heating, Refrigerating and Air Conditioning Engineers (ASHRAE) has already published appropriate guidelines, we support and encourage the use of these specific publications:

ASHRAE Standard 62-1989, "Ventilation for Acceptable Indoor Air Quality"

ASHRAE Standard 55-1992, "Thermal Environmental Conditions for Human Occupancy"

ASHRAE Standard 52-1968 (RA 76) (Reaffirmed 1976), "Method of Testing Air-Cleaning Devices Used in General Ventilation for Removing Particulate Matter"

ASHRAE Guideline 1-1989, "Commissioning of HVAC Systems"

ASHRAE Guideline 4P exists in the form of a public review draft dated 1992, "Preparation of Operating and Maintenance Documentation for Building Systems"

With the comfort of employees at stake and the prospects of increased absenteeism, plus a loss in productivity, progressive employers are finding it cost effective to ensure that indoor air quality and employee welfare are maintained. This approach is an important management tool that should be applied to both existing and planned buildings to prevent problems rather than attempting to cure them once they happen.

Adopting the building systems approach begins with adopting a ventilation standard similar to that established by ASHRAE. This standard was developed as a consensus document and was based on "real life" feedback from architects, engineers, consumer organizations, health officials, medical researchers, building owners and operators. Their experience showed that 20 cubic feet per minute of outside air per person in an office setting was effective in controlling indoor pollution. This value was chosen to control carbon dioxide and other contaminants with an adequate margin of safety and to account for health variations among people, varied activity levels, and a moderate amount of smoking.

The second focus of the building systems approach is the correct selection of air filters in commercial buildings. Again, ASHRAE has identified a suitable starting point. Filters having a minimum efficiency of 40 to 60 percent as measured by the ASHRAE 52-76 Atmospheric Dust Spot Standard should be installed. These filters should be carefully fitted and routinely serviced.

The third step is provision for regular, not less than annual, inspections of the air handling system equipment of commercial buildings to ensure cleanliness and proper operation. These inspections should include as a minimum:

- internal chambers of each air handling unit;
- coils and drain pans;
- internal insulation of chamber walls;
- humidifier sprays and reservoirs;
- internals of main air supply ducts.

Inspections should also include verification of compliance with proper schedules of filter changes and general maintenance standards of ventilation equipment.

A large body of experience has demonstrated that a building systems approach to indoor air quality is the most effective, practical and economic path for comfortable and productive indoor environment. When buildings are operated and maintained to this standard, complaints are indeed rare and smokers and nonsmokers can usually coexist in harmony.

Existing Building Stock and Ventilation Standards

The cornerstone of a successful Building Systems Approach is the use of adequate rates of ventilation for a building. The ASHRAE Standard 62-1989 is chosen because there is a growing body of evidence that confirms that buildings built and operated with this ventilation rate results in satisfied building occupants.

This specific procedure defines quantities of outdoor air of an agreed quality that must be delivered to the occupied space. Indeed, the 20 cfm/person outdoor air ventilation rate prescribed is a reversion to the ventilation rates that existed before 1973; ventilation rates specified by ASHRAE prior to the 1970's reached 30 cfm/person of outdoor air. Thus, most of the older building structures have ample capacity to operate at the 20 cfm/person rate.

A superficial look at the building stock erected in the period 1973 to 1989 suggests potential problems. During this period, minimum ventilation rates were reduced to 5 cfm/person of outdoor air. How do such buildings accommodate a Building Systems Approach?

The fact is that many of these buildings are not so badly off after all. In the first instance, design engineers who used 5 cfm per person did so using the anticipated occupancy rates of ASHRAE 62-1989 of seven persons per 1,000 ft^2. This staffing rate is high and matches the rates used to assess evacuation rates of buildings in case of fires. Therefore it reflects maximum rates of occupancy rather than typical rates. In the Department of Energy/Energy Information Administration (DOE/EIA) publication of June 1991 titled, "Commercial Buildings Characteristics 1989", the DOE estimated that the average office building has 425 ft^2 per worker yielding 2.4 persons per 1,000 ft^2. Indeed, ASHRAE 90.1P-1989 "Energy Efficient Design of New Buildings Except Low Rise Residential Buildings" uses an occupant density for use in energy calculations of 275 ft^2 per worker, yielding 3.6 persons per 1,000 ft^2. Using this average means that most buildings designed with 5 cfm are actually delivering 10 cfm/person.

Secondly, the widespread use of economizer cycles in buildings means that for extended periods throughout the year, many buildings are operating with far more than any of the minimum specified ventilation rates. Indeed, many buildings — even after the building engineer has manually closed the outside air intakes louvers — are still processing 10 to 15 cfm per person due to infiltration often brought in by the operation of exhaust fans. This was clearly illustrated in a study of a building in the City of Columbus, Ohio. When the dampers of the 38-story high rise office building were set for 5 cfm/person, the actual ventilation rate was estimated to be nearer 30 cfm/person due to massive infiltration driven by the building's exhaust fans. While this may not represent buildings specifically designed for 5 cfm/person, it does suggest that actual infiltration rates of even the so-called energy efficient buildings are considerable.

Finally, there is also accommodation in ASHRAE 62-1989 for the existing building stock. As an alternative to the Ventilation Rate Procedure, ASHRAE offers an Air Quality Procedure. This involves limiting the concentration of all known contaminants of concern to some specified acceptable levels. Thus any existing buildings can be inspected and their indoor air quality assessed and compared with the standards defined by ASHRAE. If the buildings meet these targets, and provided there is no significant level of complaints, then no further ventilation modifications are required.

New England Journal of Medicine

The press was quick to seize on an article published in a recent *New England Journal of Medicine*. This paper titled, "The Effects of Varying Levels of Outdoor Air Supply on the Symptoms of Sick Buildings" included the following:

> "Increases in the supply of outdoor air did not appear to affect workers' perceptions of their office environment or their reporting of symptoms considered typical of the Sick Building Syndrome."

The media furor that we ourselves were involved in following this statement stretched as far as radio interviews in Australia to reports in the local *Washington Post*. Both, along with many others, ran articles concluding that ventilation doesn't help sick buildings. Even BOMA's in-house newsletters published a story that ventilation was not the cause of problem buildings.

How irresponsible. We can expect ignorance from non-technical reporters, but organizations like BOMA should know better. They resisted the adoption of ASHRAE 62-1989 ventilation standards. These standards boosted ventilation rates from a minimum of 5 cfm per person to an optimum of 20 cfm per person, figures which have repeatedly been shown to work wonders in diluting pollutants and improving air quality. However, once one exceeds 20 cfm of outdoor air per person, you reach the law of diminishing returns.

In the case of the *New England Journal of Medicine* article, the researchers were comparing ventilation rates of 30 cfm moving to 64 cfm! These, they themselves admitted, were not sick buildings. This is analogous to a five foot tall non-swimmer who drowns just as effectively in six feet of water as in 60 feet of water.

Legislative Options

We believe that if you determine that regulatory action is needed to control indoor air quality, the approach should not be via source control, but by a pragmatic, effective and non-onerous series of steps to enact a Building Systems Approach herein described.

Indeed, the EPA has previously stated that poor indoor air quality costs the American economy between $10 and $12 billion each year in lost productivity. Why not champion a course of action that not only dramatically increases this country's productivity, but also helps reduce the drain on its medical resources. Remember, respiratory tract infections in this country account for $15 billion in medical care costs. Some researchers have estimated that up to fifty percent of this figure may well be associated with indoor pollutants trapped as a consequence of bad ventilation.

Surely in times of government cost-cutting and medical health care controls, there can rarely be a better vehicle to provide an option that has no association with pork-barrel politics, yet can positively achieve the stated goals while improving this country's competitiveness. This is a once-in-a-lifetime, win-win situation for all — government, managers and employees.

Gray Robertson

BUILDING SYSTEMS APPROACH

Use of appropriate design criteria

Documentation of criteria used

Ensure compliance with design specifications throughout life of building

Goal - Ensure Well-being and Comfort of Building Occupants

First Option - Discretionary Smoking

Adopt a Building System Approach, including:

- Ensure workplace conforms to ASHRAE Standard 62-1989 "Ventilation for Acceptable Indoor Air Quality"
  - Offices: Minimum 20 cfm/person of outside air
    Note: This rate accommodates a moderate amount of smoking
  - Filtration to minimum of 40 percent efficiency: ASHRAE Atmospheric Dust Spot Test

SMOKING POLICIES

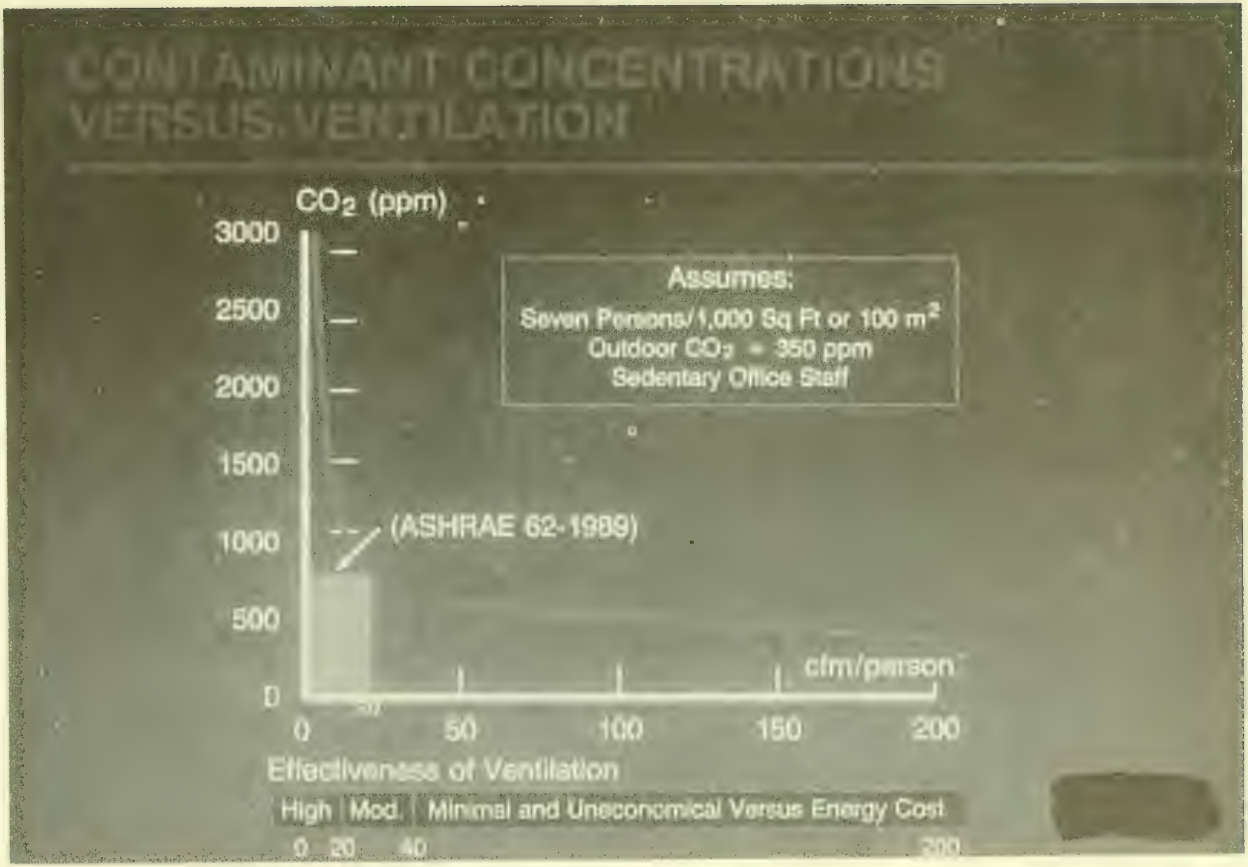
Second Option (1) - Physical Separation, No Physical Barriers

Spatial separation of smokers and nonsmokers - assuming ASHRAE 62-1989

Return air diluted by outside air and filtered before return to occupied space

Third Option - Dedicated Lounges

Local Exhaust - Principle of Operation:


CONTAMINANT CONCENTRATIONS VERSUS VENTILATION
CO_2 (ppm)
3000
2500
2000
1500
1000
500
0
Assumes:
Seven Persons/1,000 Sq Ft or 100 m²
Outdoor CO_2 = 350 ppm
Sedentary Office Staff
(ASHRAE 62-1989)
cfm/person
0
50
100
150
200
Effectiveness of Ventilation
High
Mod.
Minimal and Uneconomical Versus Energy Cost
0
20
40
200

Dedicated Lounges

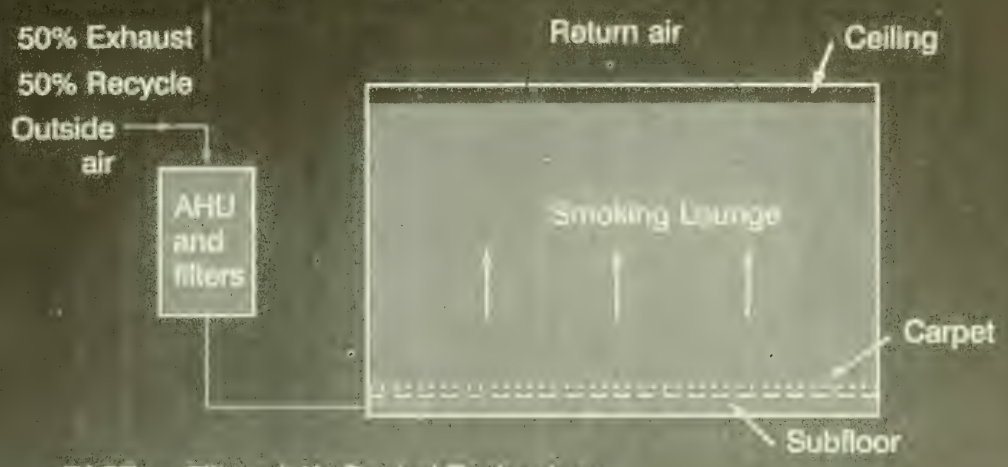
Room Size and Exhaust Capacity:

Consider:	10,000 square foot floor
Average Staff:	70
30% Smokers:	21
50% at any one time:	10
Lounge Area	150 square feet approx. (12' x 12')
150 square feet =	1.5% of 10,000 square feet
Exhaust Fan	10 x 60 cfm = 600 cfm

Cost of fan operation, 365 days/year, 24 hours/day = $50

Dedicated Lounges

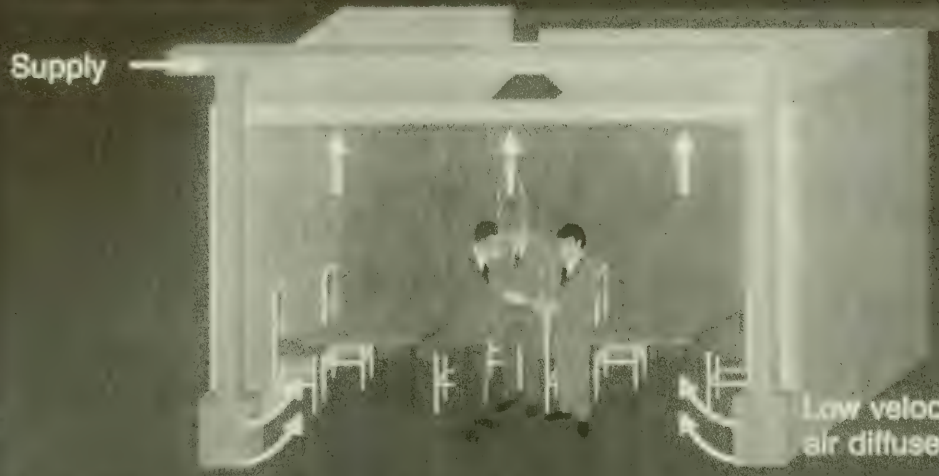
Option 3 - Displacement Ventilation (F.A.C.T.)



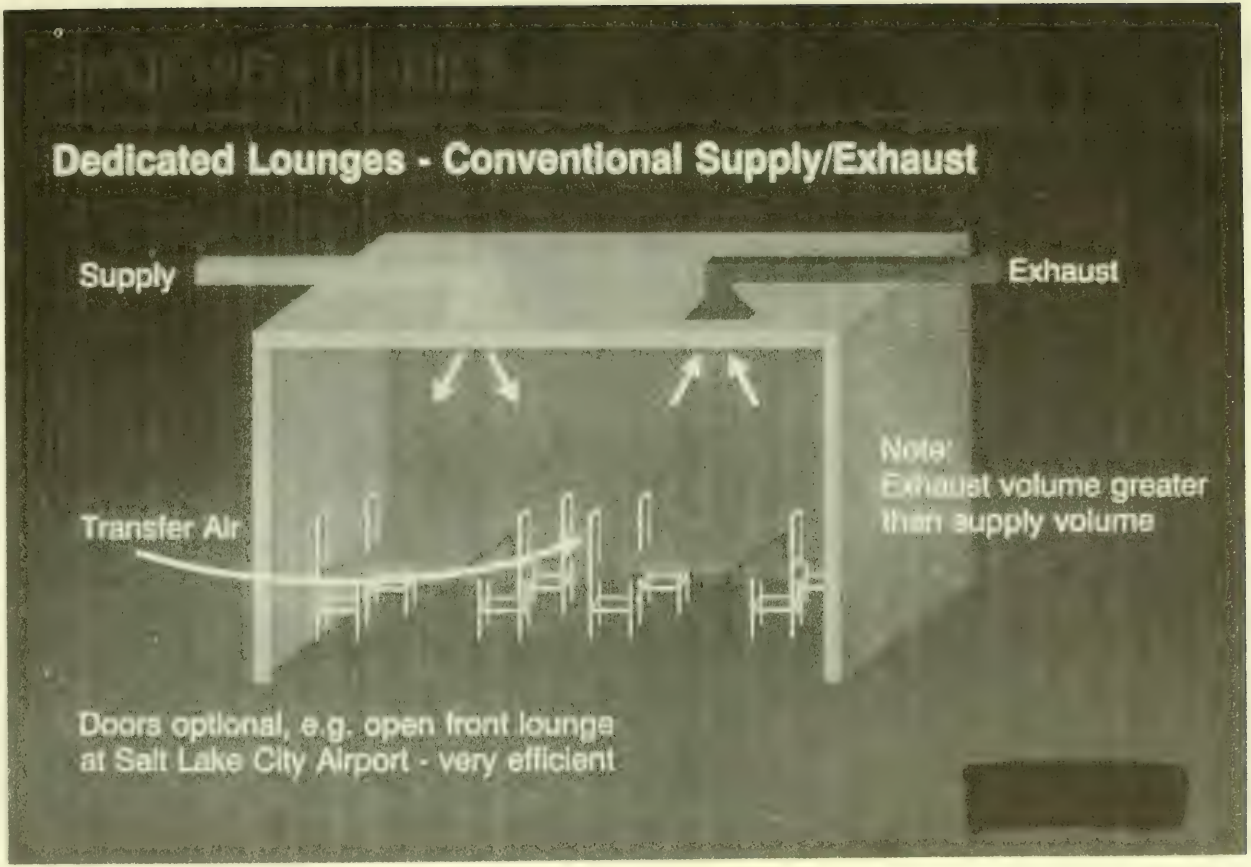
FACT = Filtered Air Control Technology

(Philip Morris Company)

Dedicated Lounges - Displacement Ventilation



Air supply at foot level, say 65-68°F
Air exhaust at ceiling, say 73-75°F


Dedicated Lounges - Conventional Supply/Exhaust
Supply
Exhaust
Transfer Air
Note:
Exhaust volume greater
than supply volume
Doors optional, e.g. open front lounge
at Salt Lake City Airport - very efficient

Example - Benzene

OSHA	PEL	1 ppm (TWA 8 hr)
ACGIH	TLV	10 ppm (TWA 8 hr)

Offices	9	39	0.0025	0.0020
Homes	7	820	0.0042	0.0025
Transport	3	383	0.0024	0.0092

Benzene concentration: Transport greater than Homes, Homes greater than Offices

RESPIRATORY DISEASES AND ABSENTEEISM (1)

- Respiratory Tract Infections Are the Most Common Infectious Illnesses Among Humans
- Estimated to Annually Cause in the United States:

  150 Million Lost Workdays

  75 Million Physician Visits

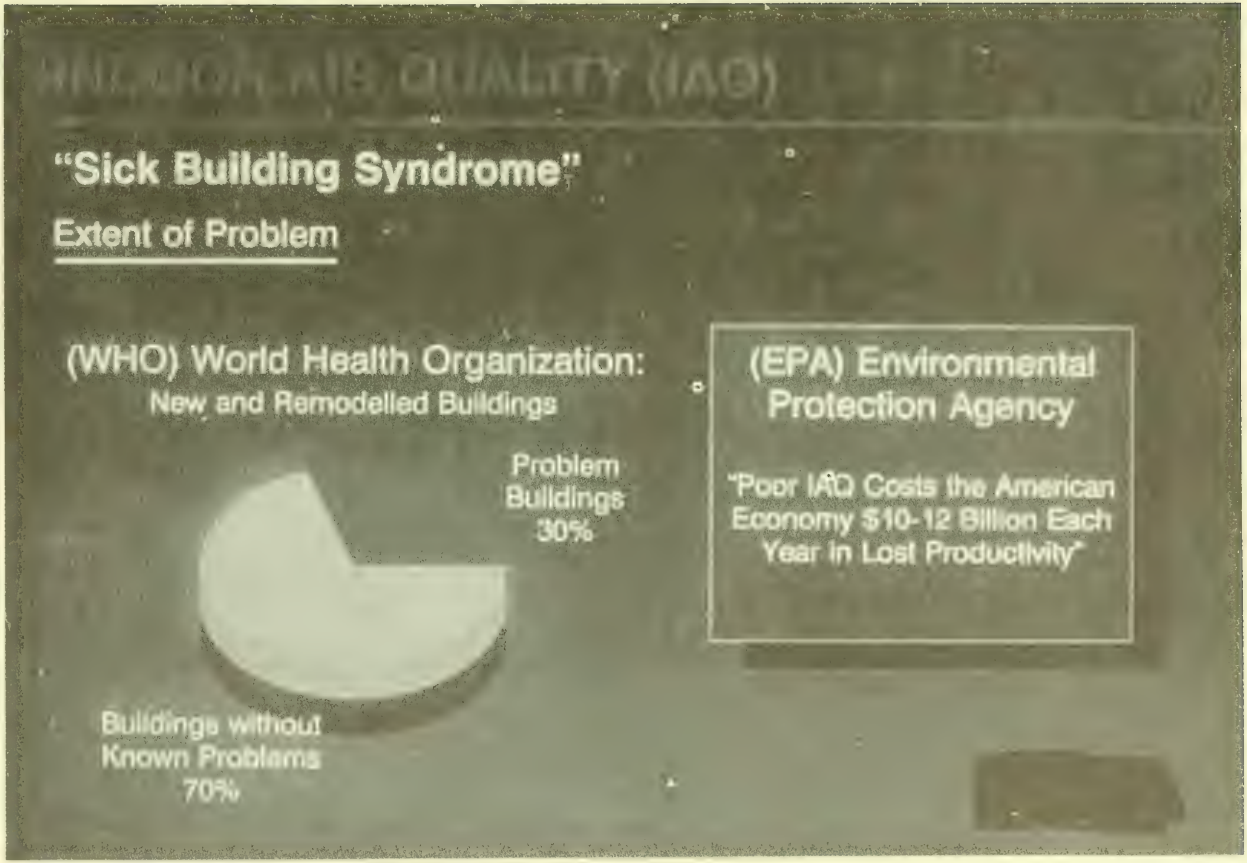
  $15 Billion Medical Care Costs

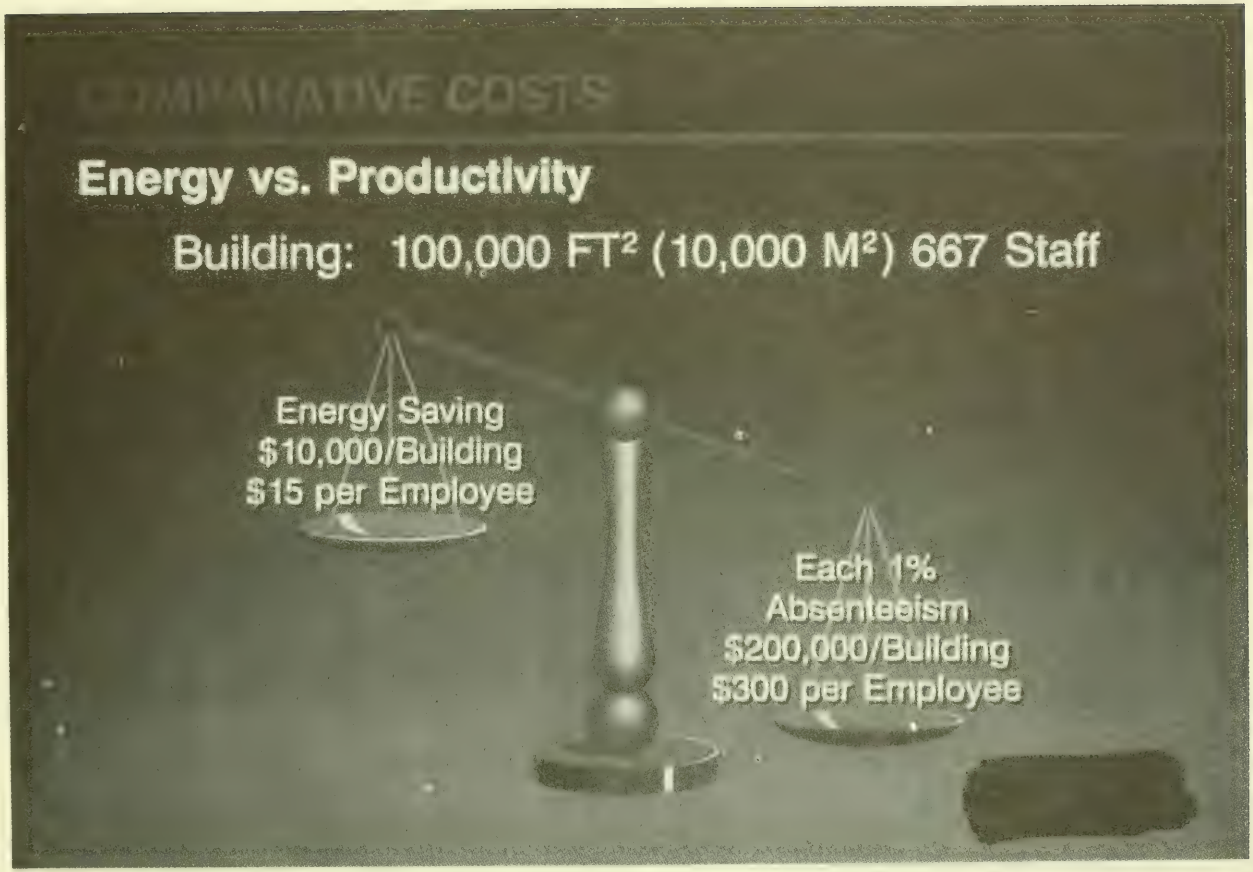
Source: Garibaldi & Dixon
Am. Journal Med. 1985

RESPIRATORY DISEASES AND ABSENTEEISM (2)

- *Effects of Tight Buildings:*
  Walter Reed Army Inst.
  4 Year Study (1983 - 86)
- Approximately 400,000 Recruits, Housed in
  50% Energy Efficient Buildings
  50% Non-Energy Efficient Buildings
- Results: Rates of Acute Respiratory Diseases Ranged from [illegible] Higher in the Tight Buildings

Source: Journal American Med. Assoc. 1988


"Sick Building Syndrome"
Extent of Problem
(WHO) World Health Organization:
New and Remodelled Buildings
Problem Buildings 30%
Buildings without Known Problems 70%
(EPA) Environmental Protection Agency
"Poor IAQ Costs the American Economy $10-12 Billion Each Year in Lost Productivity"


COMPARATIVE COSTS
Energy vs. Productivity
Building: 100,000 FT² (10,000 M²) 667 Staff
Energy Saving
$10,000/Building
$15 per Employee
Each 1%
Absenteeism
$200,000/Building
$300 per Employee

Statement of

Lawrence W. Rogers, Director

Office of Workers' Compensation Programs

United States Department of Labor

before the

Subcommittee on Public Buildings and Grounds

Committee on Public Works and Transportation

United States House of Representatives

April 22, 1993

Mr. Chairman and Members of the Subcommittee:

Good morning. I am the director of the Office of Workers' Compensation Programs (OWCP), a part of the Employment Standards Administration. OWCP administers the Federal Employees' Compensation Act (FECA), which is the workers' compensation law covering all federal employees. I appreciate this opportunity to discuss with you how the FECA covers claims for injuries arising from work-place exposure to environmental tobacco smoke (ETS).

I understand the Committee is interested in the responsibility of the government for injuries that may arise out of exposure to tobacco smoke in public buildings. I will discuss this issue

from the aspect of the government as an employer, and will explain how injuries or illnesses sustained by employees that arise out of work-place exposure, including to environmental tobacco smoke (ETS), are compensated. I want to emphasize that the FECA already covers ETS-related injuries and illnesses, like any other work-related illness, and will continue to do so as it is not affected by the outcome of the legislation being considered. In addition, since such injuries are covered, the FECA's exclusive remedy provision bars any attempt by employees or their families to recover damages from the government through any other means.

Before going into details on coverage, let me first provide some general background on the FECA. I will then turn to the specific issue of claims arising out of environmental tobacco smoke exposure.

The FECA provides a variety of benefits to those Federal employees who are injured on the job, and to the families of employees who die as a result of work-related factors. In 1991, we marked the seventy-fifth anniversary of the FECA, which covers more than three million civilian employees of the United States in seventy-seven government agencies around the world. Certain other individuals are also covered, including some volunteers. When an employee alleges an injury due to the job, a claim is filed with the employing agency, which processes it and sends it

to one of the twelve OWCP district offices throughout the country for adjudication. An OWCP claims examiner evaluates the claim, may undertake or direct evidentiary development such as medical examinations and air quality studies, and finally makes an eligibility decision on the basis of the evidence. If the claim is accepted, appropriate benefits are paid. If the claim is denied, the claimant has a variety of administrative review and appeal options.

The benefits available under the FECA include payment of all medical expenses resulting from a work-related injury or occupational illness as well as wage-loss benefits for as long as the disability continues. Compensation for the wage loss is paid at two-thirds of the worker's salary, or three-fourths if there is at least one dependent. Death benefits available to the survivors of employees who die of work-related injuries or illnesses, provide 50 percent of the salary to a surviving spouse, and up to 75 percent if there are two or more dependent children. Another benefit is the schedule award, a payment for permanent loss or loss of use of a part of the body, such as a leg, an eye or a lung.

All FECA benefits paid last year totalled about $1.8 billion. The money is paid from the Employees' Compensation Fund, which is administered by OWCP and is replenished by billing each employing agency through a chargeback system. The appropriated fund

agencies meet their chargeback costs through their annual budget requests to Congress. Costs which are not billable to an agency are paid out of the Department of Labor's appropriation.

There is no limit on the condition which may be covered by the FECA, although those which arise from an employee's willful misconduct, intoxication, or intentional injury are excluded from coverage. Claims fall into two general categories: 1) traumatic injuries, which constitute about 90 percent of all claims, are those caused by a discrete event which occurs during one work shift; and 2) occupational diseases, which develop over time because of repeated exposure, stress or strain, as for example those which arise from repeated exposure to a toxic agent.

In either traumatic injuries or occupational disease claims, the claimant has the initial burden of proving the basic elements of the claim. The elements which are most relevant when we talk about work place exposure to environmental tobacco smoke are:

- 1) **Fact of injury** - meaning the employee must show that an event or exposure actually occurred which had some impact on the individual requiring medical treatment or resulting in disability;

- 2) **Performance of duty** - requiring that the exposure occurred in the performance of the job, or otherwise as a result of a factor of employment; and

- 3) **Causal relationship** - that is, that the medical condition for which compensation is claimed was caused or aggravated by the work injury or event.

The degree of proof needed varies for each case. In some straightforward traumatic injury cases, such as a broken leg resulting from a fall at work, these factors may be relatively easy to show. In more complex occupational disease cases, such as those which generally result from environmental exposures, these elements may be more difficult to establish. This is so in part because the necessary medical evidence may be more difficult to obtain. Particularly when it comes to establishing a causal relationship between the claimed condition and the exposure at work, the evidence must be objective, well-rationalized and based on scientific principles. I will return to this point a little later, in the specific context of environmental tobacco smoke.

Now that I have given some background on the FECA and how the claims process works, let me now turn to FECA coverage of environmental tobacco smoke. First, FECA claims have been filed due to work place injuries from environmental tobacco smoke and

such claims have been accepted and compensation paid. Although OWCP's data base is not indexed in specific enough detail to identify all claims arising from exposure to tobacco smoke, we have identified a number of cases in which claims for ETS-related injuries were accepted. Although we do not know exactly how many ETS-related claims have been filed, we believe the number of cases involved to date has been relatively small.

The claims have been for both traumatic and occupational diseases, and the medical conditions resulting from ETS exposure which have been accepted include aggravation of asthma, rhinitis, non-specific lung disease, allergic reactions (resulting in coughing, hoarseness and swelling of the airways), and hypersensitivity to tobacco smoke. (We can find no record, however, of having received or approved a claim of lung cancer as a result of tobacco smoke.) Injuries have disabled individuals to varying degrees. Many of the approved claims involved only medical costs or very short periods of disability. Others were for temporary aggravation of conditions such as asthma, which the individuals already had but which were exacerbated for short periods. Other cases, however, involve long-term disability; we have identified claims in which the employee has been totally disabled for several years, and compensation and medical benefits paid total thousands of dollars.

It is clear from our review of FECA claims involving ETS-related injuries that the system has shown itself to be able to elicit and interpret the necessary medical evidence in regard to the adjudication of claims, and provide the appropriate medical and other benefits. It is also clear that the ETS-related injuries have resulted in direct costs to the government. While I am not here to speak to the merits of the proposed legislation to ban smoking in all government buildings, the proposed legislation would not change the responsibility of the government, in its role as an employer, for ETS-related injuries. Nor should the growth in scientific evidence concerning environmental tobacco smoke, such as set forth in the recent testimony before this committee by the Surgeon General and by the Director of the Office of Health and Environmental Assessment of the Environmental Protection Agency, have a significant impact on the FECA adjudication process or necessarily result in the acceptance of a large number of additional claims. As illustrated by the cases I mentioned earlier, there has been (for Federal workers' compensation purposes) a sufficient medical basis to causally relate environmental tobacco smoke to individual medical conditions.

I would add that only as far as the proposed legislation and the new scientific findings raise the consciousness of employees regarding the hazards of environmental tobacco smoke, it is possible that we may see an increase in the number of claims

filed. Moreover, in some cases where evidence has not previously been available to establish a causal connection between a particular condition and tobacco smoke, the new scientific studies may make it easier for claimants to support some claims for individual medical conditions. This may be especially common for persons with conditions that have a long latency period. Further, it is possible that, over time, claims would decrease, because of the lack of smoke to which workers would be exposed. I want to emphasize that we have no systematic basis for these conclusions, however, but draw them from anecdotal observations of the process as it now exists.

I appreciate this opportunity to tell you about OWCP and its handling of claims for environmental tobacco smoke related injuries by Federal employees. I will be happy to answer any questions you may have.

FACTSHEET
CONSTRUCTION AND ENERGY COSTS
OF SEPARATELY VENTILATED SMOKING ROOMS
IN GSA-OWNED AND -LEASED FEDERAL BUILDINGS

ISSUE: To estimate the construction cost and energy consumption needed to establish separately ventilated smoking rooms in GSA-owned and -leased Federal buildings.

GIVEN: Currently GSA owns or leases 7800 buildings nationwide. The average utility cost is approximately $1.00 per SF.

ASSUMPTIONS:

1) Space required is approximately 250 SF per location.
2) Cost of construction is $30.00 to $50.00 per SF.
3) Depending on climate, the energy cost for smoking rooms would double or triple.
4) An average of one such room per location. (Actual may vary from none to several.)
5) Cost of replacing space used for smoking rooms is approximately $16 per sq ft.

DISCUSSION:

1) Space required:
@ 250 SF in each of 7800 buildings

250 sq ft * 7800 buildings = 1,950,000 sq ft of space required

2) Cost of Construction:
@ $30.00 to $50.00 per SF

1,950,000 sq ft x $30.00 = $58.5 million

1,950,000 sq ft x $50.00 = $97.5 million

3) Cost of Energy:
@ double or triple current cost of $1.00 per SF

(@double =) 1,950,000 sq ft x $1.00 = $1.95 million additional cost annually

(@triple =) 1,950,000 sq ft x $2.00 = $3.9 million additional cost annually

4) Cost to tenant agencies of replacing 1,950,000 sq ft of space used for smoking rooms

$ 16 per sq ft x 1,950,000 sq ft = $31.2 million

CONCLUSION: The cost to establish separately ventilated smoking rooms in the 7800 GSA-owned and leased facilities would range from $58.5 million to $97.5 million, and energy cost could increase by $1.95 to $3.9 million annually. The replacement of space used for smoking rooms will cost the Federal government $31.2 million annually.

CONTACT: James F. Steele, Jr.
Assistant Commissioner
Office of Real Property Management and Safety

Statement of
David Zeigler
Acting Assistant Secretary
Occupational Safety and Health Administration
U.S. Department of Labor
Before the
Subcommittee on Public Buildings and Grounds
Committee on Public Works and Transportation
U.S. House of Representatives

April 22, 1993

Mr. Chairman and Members of the Subcommittee:

Thank you for this opportunity to discuss H.R. 881, the "Ban on Smoking in Federal Buildings Act." First, let me say that the Administration generally supports your efforts to protect the health and safety of Federal workers. Those efforts are consistent with the mission of the Occupational Safety and Health Administration (OSHA), which Congress charged in 1970 with the responsibility for assuring "so far as possible every working man and woman in the Nation safe and healthful working conditions..."

As you know, the scientific and medical communities have long recognized the potential adverse health effects associated with smoking. Concern about the negative effects of smoking has increased in recent years, with scientific research turning to the potential risks that second-hand tobacco smoke poses to nonsmokers. A wide range of health effects caused by second-hand tobacco smoke has been reported by the Surgeon General, the National Research Council, the Environmental Protection Agency (EPA), the National Institute for Occupational Safety and Health (NIOSH) and private researchers. These effects include acute annoyance, eye and respiratory tract irritation, low birthweight,

the development of chronic pulmonary diseases, cardiovascular disease, and lung cancer. In addition, NIOSH has determined that second-hand tobacco smoke is a potential occupational carcinogen and possible heart disease risk factor in nonsmoking workers, and both the Surgeon General, and EPA in its newly released report, have classified second-hand tobacco smoke as a known human (Group A) carcinogen.

Clearly, therefore, H.R. 881 addresses a serious health issue--one which OSHA also has under very active review. In order to place my comments on H.R. 881 in perspective, I would like to briefly discuss OSHA's statutory authority to prescribe and enforce workplace safety and health standards; the agency's role with regard to Federal employees' safety and health; and the agency's specific efforts to protect workers from the hazards of indoor air pollution, including second-hand tobacco smoke.

OSHA's Authority

First I will briefly explain OSHA's authority, under the Occupational Safety and Health Act of 1970 (OSH Act), to prescribe workplace standards to regulate employees' safety and health. Section 6(b) of the OSH Act charges OSHA with setting occupational safety and health standards when there is significant risk of injury or material impairment of health.

In order to show that a workplace condition poses a material impairment of health, OSHA relies on research and technical support from other agencies, such as NIOSH (OSHA's sister agency

in the Department of Health and Human Services, created by the OSH Act to conduct research and training and make recommendations regarding occupational safety and health standards), EPA, and on the private sector. OSHA may begin a rulemaking when reliable technical information to support the rule comes to its attention or when petitioned by a party.

The occupational safety and health protections provided under the OSH Act extend to private sector employees at a virtually unlimited variety of worksites. The OSH Act also provides for the safeguarding of Federal employees' safety and health through a requirement that Federal agencies comply with standards consistent with those OSHA issues for private sector employers.

Federal Employee Safety and Health Protection

With regard to Federal employees' safety and health, section 19 of the OSH Act places responsibility for their protection with the head of each Federal Agency. Executive Order 12196, which has been in effect since February 1980, defines OSHA's role and the safety and health duties of the Executive departments and agencies. The Executive Order and OSHA's implementing regulations require each agency to operate an occupational safety and health program in accordance with the principles and basic program elements issued by OSHA. Although the agencies must operate a program in accordance with OSHA's requirements, they

are permitted enough flexibility to implement their programs in a manner consistent with their size and respective missions.

Federal agencies must comply with all OSHA standards for the private sector unless OSHA approves alternate standards. The agencies are required to conduct inspections of their workplaces, at least annually, respond to employee reports of imminent danger or serious hazards, and assure that unsafe conditions are abated promptly. Each agency is also required to compile, maintain, and analyze statistics on occupational injuries and illnesses. A report on each agency's occupational safety and health program is submitted to OSHA each year.

The Executive Order also assigns a role to the General Services Administration (GSA), which provides workspace for more than one million Federal employees. As an element of GSA's property management responsibilities, the Executive Order requires GSA to assure prompt attention to reports of unsafe or unhealthful conditions in facilities subject to their authority.

OSHA's Responsibilities in the Federal Sector

OSHA is given a number of responsibilities under Executive Order 12196. The agency is required to provide oversight, leadership and assistance to all Federal agencies as they carry out their safety and health responsibilities, including assistance with training materials, technical services, and programs for information exchange. Evaluation of other agencies' safety and health programs, particularly those of larger

agencies, is an important aspect of OSHA's oversight responsibility. OSHA is further required to submit to the President a summary report of the status of Federal agency safety and health each year.

As part of its oversight of Federal agencies' programs, OSHA conducts inspections in Federal agencies. In FY 1992, OSHA conducted more than 1,070 inspections in Federal agencies and found more than 4,200 violations. In the overwhelming majority of cases, we find that Federal agencies cooperate to abate workplace hazards expeditiously. In fact, Federal agencies fail to abate only 3 percent of all violations in a timely fashion.

OSHA ACTION ON INDOOR AIR QUALITY

I will now turn to a discussion of OSHA's initiatives to address the overall issue of indoor air quality. The agency's past efforts have focused on providing information and guidance to its field personnel to be used in the evaluation of indoor air quality conditions in the Nation's workplaces.

Technical Assistance and Consultation

OSHA also encourages safe and healthful working environments through a variety of methods in addition to the enforcement program. For instance, the agency's field offices provide technical assistance and offer a wide variety of written publications and other information on occupational safety and health to employers and employees. OSHA's consultation service

provides assistance, either at the employer's establishment or off site, on all aspects of occupational safety and health. Consultants with expertise in occupational health are available to help employers find ways of eliminating hazards associated with indoor air contaminants.

Request For Information on Indoor Air Quality

While OSHA's past efforts to address indoor air quality problems have been somewhat limited, the agency recognizes the seriousness of the issue, and is now going beyond these initial efforts to review the need for rulemaking to address indoor air quality and second-hand tobacco smoke.

In 1987, OSHA received several petitions requesting an emergency temporary standard (ETS) for second-hand tobacco smoke. The statutory requirement which OSHA must meet to issue an ETS is greater than for a normal rulemaking, requiring a finding that employees are exposed to a "grave danger." OSHA denied the petitions for an ETS based on the agency's determination that data available at that time did not demonstrate the existence of a "grave danger" due to workplace exposure to second-hand tobacco smoke, within the meaning of Section 6(c) of the OSH Act. Following the denial, one petitioner filed suit with the U.S. Court of Appeals asking for a review of OSHA's decision. The court upheld OSHA's decision.

Our determination that there was not sufficient evidence for an ETS did not, however, mean that OSHA felt there was not a

hazard. OSHA proceeded in September 1991 to publish a Request For Information (RFI) in the Federal Register to obtain information on indoor air conditions. The RFI covers such issues as health effects of contaminants, the operation of ventilation systems, building maintenance programs in use, and the hazards of specific contaminants such as second-hand tobacco smoke and radon. OSHA is currently reviewing more than 1,200 comments submitted in response to the request in order to determine the appropriateness and feasibility of an indoor air standard.

Key issues addressed in the public comments concern ventilation performance standards, worker training on the operation and maintenance of Heating, Ventilation and Air Conditioning (HVAC) systems, pollution source control, and technical guidance. The specific pollutants addressed in the comments include second-hand tobacco smoke, bioaerosols, carbon monoxide, and asbestos, among others.

Following the issuance of the RFI, OSHA received several more petitions requesting that the agency promulgate a comprehensive indoor air standard, and that it separate second-hand tobacco smoke from the larger indoor air quality issue, and again consider issuing an ETS.

The issues raised by these petitions are currently under active consideration.

Interagency Activities

OSHA is also working closely with many other agencies who have a role in various aspects of indoor air quality, including the Department of Health and Human Services, NIOSH, EPA, GSA and others. OSHA is a co-chair of the Interagency Committee on Indoor Air Quality, which coordinates the efforts of the Federal Government, and represents the Department of Labor on the Surgeon General's Interagency Committee on smoking.

As part of this interagency cooperation, EPA's recent report on "*Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders*" has been included in the public docket for OSHA's RFI on indoor air quality. EPA's report classified second-hand tobacco smoke as a known human (Group A) carcinogen and concluded that it is responsible for approximately 3,000 excess lung cancer deaths a year in nonsmoking adults. Of these 3,000, EPA estimates that 2,200 are attributable to nonhome exposures (combined workplace and social settings). OSHA views this as a significant study which was developed using a credible methodology, and it will be considered by the agency, along with all other comments, in order to determine further agency action.

Comments on H.R. 881

With regard to H.R. 881, OSHA supports the objective of the bill, to protect Federal employees and the public from the

hazards posed by second-hand tobacco smoke in Federal buildings. The overall intent of the legislation is consistent with OSHA's mission to assure safe and healthful working conditions.

Mr. Chairman, we appreciate your efforts to focus public discussion on this important issue. Thank you again for the opportunity to testify on H.R. 881. I will be happy to answer any questions you may have.

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